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The American Heart Journal

VOL. VII AUGUST, 1932 No. 6

Original Communications

THE INCIDENCE AND SIGNIFICANCE OF THE DEEP Q-WAVE IN LEAD III OF THE ELECTROCARDIOGRAM*

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PHILADELPHIA, PA.

IN 1926, Wilson[1] stated that a deep Q-wave in Lead III is an important electrocardiographic finding following coronary occlusion. Parkinson and Bedford[2] and Levine[3] also noted this wave in some of their cases of coronary occlusion. Pardee's work,[4] however, was the first to arouse widespread interest in the Q-3 wave. His contributions include (a) the proposal of criteria to separate significant from nonsignificant Q-3 waves, and (b) a study of the incidence of presumably significant Q-3 waves in clinical material. His conclusions include the following statements:

"The finding of a large Q-3 indicates disease of the left ventricle, so that the right ventricle predominates during the spreading of the contraction in spite of the left axis deviation or normal axis."

"The majority of such records are obtained from patients with the anginal syndrome, but certain patients with myocardial fibrosis and congestive failure, certain patients with rheumatic heart disease, especially with pericarditis, and a few with hypertension will give such records. Certain patients who have cardiac symptoms but no definite evidence of cardiac disease have been found to show this large Q-3."

"The occasional finding of a large Q-3 in normal hearts may be due to an unusual distribution of the branches of the A-V bundle, and a high position of the diaphragm may be a contributing factor."

In this paper, the material at our disposal has been analyzed in an attempt to evaluate the importance of Q-3. We have adhered to the criteria for a significant Q-3 proposed by Pardee.

Our cases have been divided arbitrarily as follows:

I. A. Seven hundred and nine presumably normal college students.
B. One hundred and seventeen college athletes.

*From the Edward B. Robinette Foundation, Medical Clinic, Hospital of the University of Pennsylvania.

II. Five hundred cases from the wards and cardiovascular section. The only criteria for inclusion in this group included (1) adequate records, (2) unquestionable evidence of cardiovascular disease.

III. Nineteen hundred unselected electrocardiograms from the files of the Cardiographic Laboratory.

IV. One hundred and sixteen cases having the anginal syndrome.

V. One hundred and forty-five corporation executives.*

VI. Twenty-five pregnant women.

There is no overlapping among Groups I, III and VI. Cases in Groups II, IV and V which happened to be studied within the time period of the material in Group III are included.†

Group I.—The records of 709 apparently normal college students were made available by Dr. Francis Clark Wood. In this group, not a single significant Q-3 was found. There were nine tracings in which the amplitude of a Q-3 was sufficient to satisfy Pardee's requirement in this respect, but they did not fulfill certain of his other criteria, since all showed either right axis deviation or a tendency toward this deviation.

The records of one hundred and seventeen athletes of the University of Pennsylvania were also made available by Dr. Wood. This group included members of the crew, the football, soccer and lacrosse teams. One significant Q-3 wave was found. The recorded physical examination of this individual disclosed a notation of systolic and diastolic murmurs at the apex. Through an error in filing this record had been placed in the normal group. Another tracing showed a Q-3 wave of sufficient depth, but there was a tendency toward right axis deviation.

Group II.—The electrocardiograms of 500 patients with definite cardiovascular disease were examined. This group included the usual types of patients seen in the ward and the out-patient cardiovascular clinic.

There were 31 (6 per cent) significant Q-3 waves in this group. The etiological diagnosis of the patients showing a significant Q-3 wave were:

I. Rheumatic type, 3 cases: (a) mitral stenosis, 1; (b) mitral stenosis and aortic insufficiency, 2.

II. Hypertension and/or arteriosclerosis, 15 cases.

III. Syphilis, 8 cases: (a) aortitis, 5; (b) aortitis and aortic insufficiency, 2; (c) aneurysm, 1.

IV. Thyrotoxic heart, 1 case.

V. Etiology undetermined, 4 cases.

The youngest patient showing a significant Q-3 was thirty-two years of age, the oldest seventy-two. Five cases (16 per cent) were found to

*The data in this group were obtained from routine health examinations by Dr. T. Grier Miller and one of us.

†Group III represents a cross section of material from all sources subjected to electrocardiographic study in this Clinic. Time periods were chosen at random and in each period tracings were taken consecutively. No more than one tracing of any one patient was included.

have the anginal syndrome. In two cases, one arteriosclerotic and the other of unknown etiology, the hearts were transversely inclined by a high diaphragm, and both showed definite evidence of cardiovascular disease.

The electrocardiograms in 18 of the 31 cases with significant Q-3 waves were otherwise normal; 8 showed T-wave changes; 4 slurred QRS complexes; and 1, complete heart-block.

Group III.—Nineteen hundred unselected electrocardiograms taken from the files were examined in an attempt to determine the general prevalence of a significant Q-3 wave among the types of material ordinarily studied in our cardiographic laboratory. In contrast to Group II, which consisted of ward and clinic patients, this group also included private patients. There were 78 (4.1 per cent) significant Q-3 waves in this group. Of these, 40 (5.2 per cent) were found among 768 private patients and 38 (3.44 per cent) among 1132 ward and clinic patients. The clinical classification of this group is shown in Table I.

Of the 75 patients whose records were available, or concerning whom information was obtainable from the referring physician, 63 (84 per cent) could be definitely classified as having cardiovascular disease, and of this number, 22 (41.5 per cent) had a history of either angina pectoris or coronary occlusion, or both.

TABLE I

CLASSIFICATION OF THE 78 CASES FROM GROUP III EXHIBITING A DEEP Q-3 WAVE

	NUMBER	ANGINAL SYNDROME		HYPERTENSION AND/OR ARTERIOSCLEROSIS*	RHEUMATIC TYPE	CARDIOVASCULAR SYPHILIS	HYPERTHYROIDISM	MYOCARDIAL DISEASE ETIOLOGY UNKNOWN	CARDIOVASCULAR DISEASE DOUBTFUL	EVIDENCE OF CARDIOVASCULAR DISEASE, NEGATIVE OR INSIGNIFICANT	NO RECORDS AVAILABLE EXCEPT ELECTROCARDIOGRAM
		ANGINA PECTORIS	CORONARY OCCLUSION								
Private	40	12	6	8	1	0	0	3	4	3	3
Ward and clinic	38	2	2	11	6	3	4	5	1	4	0
Total	78	14	8	19	7	3	4	8	5	7	3

*Without the anginal syndrome.

In this group, the marked predominance of angina pectoris and coronary occlusion in the private cases as contrasted with the ward and clinic cases is striking. It is noteworthy that rheumatism, syphilis and goiter were found more frequently among the ward and clinic cases.

TABLE II

CLINICAL DATA IN THOSE CASES EXHIBITING A DEEP Q-3 WAVE WHICH WERE CLASSED IN TABLE I AS NEGATIVE OR DOUBTFUL

NEGATIVE OR INSIGNIFICANT FINDINGS		
AGE	CARDIAC SYMPTOMS AND FINDINGS	COMMENTS
73	No cardiovascular symptoms; no positive physical findings except extrasystoles. Heart normal in size.	Two complete examinations more than a year apart.
58	Complaint of dyspnea on exertion. Extrasystoles.	Has been under observation two years.
32	A physician apprehensive about extrasystoles. No other symptoms; no positive physical signs except extrasystoles. Small T-wave in Lead II. Heart transversely placed but of normal size.	Has had extrasystoles since he was a medical student eight years ago. The small T-2 was regarded as due, in all probability to the position of the heart.
59	No cardiovascular symptoms. No positive physical findings.	Renal glycosuria. Has been under observation of various physicians for many years.
41	Numerous and varied complaints, including precordial pain, radiating down left arm. No positive physical findings except obesity.	Complete studies made in medical ward.

EVIDENCE OF CARDIOVASCULAR DAMAGE INCONCLUSIVE		
AGE	CARDIAC SYMPTOMS AND FINDINGS	COMMENTS
ABOUT 50	Precordial pain; low T-waves in Lead II.	Diabetes mellitus. Moderate anemia. The precordial pain was not anginal in type.
60	History not obtainable. T-waves low in all leads. Extrasystoles. Orthodiagram shows slightly enlarged heart.	
43	Tachycardia observed during convalescence from alcoholic debauch. No other cardiovascular findings.	Died shortly after this study was made in another bout of alcoholism.
38	Obesity; palpitation, precordial discomfort, dyspnea on exertion. X-ray picture shows slight generalized enlargement of heart. Low T-wave in Lead II.	Highly neurotic, although findings suggest cardiovascular disease.
40	History of dyspnea on exertion, weakness, dizziness, swelling of ankles. These came on in association with attack of arthritis.	Negative on reexamination, but electrocardiogram no longer showed significant Q-3 wave.
27	History of precordial pain, palpitation, dyspnea, edema of ankles. No positive physical findings.	
44	Tachycardia and slight cyanosis.	Negative on reexamination but electrocardiogram no longer showed significant Q-3 wave.

The diagnosis of the twelve cases in the groups considered negative or doubtful were as follows: extrasystoles only—4, cardiac neurosis—2, diabetes mellitus—1, alcoholism—1, renal glycosuria—1, undiagnosed—3.

The data of possible significance from the cardiovascular viewpoint is listed in Table II. These cases presented diagnostic difficulties and the classification is arbitrary. Cases were classed as negative when none of the available evidence seemed to point to cardiovascular disease. There were five cases in this group. Of these, three were elderly (fifty-eight, fifty-nine and seventy-three years of age). The possibility must be conceded that cardiac damage, which we were unable to detect, was present. The other two patients were forty-one and twenty-six years of age; the former was obese, the latter had a transversely placed heart and his electrocardiogram showed numerous extrasystoles.

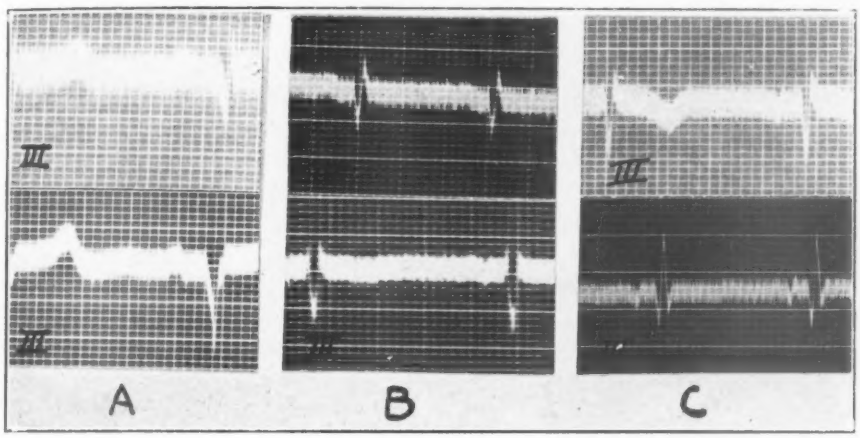

Fig. 1.—Variability of significant Q-3 waves. In each case a period of several months elapsed between the first and second tracings. *A* and *B* are from cases with the anginal syndrome; *C* (upper tracing) is from a pregnant woman; the lower tracing was taken several months after delivery.

Seven cases were classified as doubtful because their complaints or findings pointed to cardiac damage but sufficient evidence was not found to warrant a definite diagnosis.

In this entire group of 78 cases with significant Q-3 waves the electrocardiograms were otherwise normal in 38 cases; there were T-wave changes in 32; the QRS complexes were slurred in four; and four cases showed various degrees of heart-block.

Group IV.—The electrocardiograms of one hundred and sixteen cases with the anginal syndrome (angina pectoris or coronary occlusion or both) were examined. There were 31 cases with significant Q-3 waves (26.7 per cent). The majority (62 per cent of the entire group) showed a left axis deviation, the QRS complex of Lead III in many showing (1) a small upright wave followed by a deeply inverted wave and occasionally by another upright wave; (2) a single inverted wave; or (3) an in-

verted wave followed by an upright wave. Tracings repeated later in some of these patients showed changes in the QRS complexes, which although slight, were sufficient according to Pardee's criteria, to compel a reclassification. Some developed significant Q-3 waves and others lost them (Fig. 1, *A* and *B*).

Among the 31 cases with significant Q-3 waves, the electrocardiograms were normal in other respects in eight cases; T-wave changes were present in 22 cases. One showed numerous ventricular extrasystoles.

Group V.—The electrocardiograms of 145 corporation executives were studied. This material was obtained from a health survey made by Dr. T. Grier Miller and one of us (C. C. W.). The cases studied comprised practically all of the executives stationed in a single geographic unit of one corporation. The data were considered to have some value as a control particularly for Group IV. None of these corporation executives appeared to be suffering from the anginal syndrome (although one has since developed coronary occlusion); nevertheless their age incidence was comparable to that of sufferers from the anginal syndrome. Furthermore, they represent a class from which many victims of angina pectoris are recruited, namely business men carrying heavy responsibilities.

There were eight significant Q-3 waves in this group. The clinical and fluoroscopic data of the eight cases were then studied and seven showed evidence of heart disease. In the eighth individual, a man forty-seven years of age, repeated studies over the course of three years have failed to reveal any evidence of cardiovascular disease.

The electrocardiograms showing a significant Q-3 wave were otherwise normal in three cases; two showed T-wave changes and three other abnormalities.

Group VI.—In order to determine whether a high diaphragm favors the production of a significant Q-3 wave, electrocardiograms of twenty-five pregnant women (ninth month) were made. In evaluating the findings in this group, cognizance must be taken of the fact that other factors besides elevation of the diaphragm influence the heart during pregnancy.

There were four deep Q-3 waves but only 3 (12 per cent) were considered significant. The fourth was found in a tracing which also showed a right axis deviation.

Several months after delivery, electrocardiograms were repeated on two of the three patients; in both instances there was a disappearance of the significant Q-3 wave (Fig. 1, *C*).

DISCUSSION

Clinical Importance.—The finding of but one significant Q-3 wave among the electrocardiograms of 826 college students with presumably normal cardiovascular systems reflects the rarity of this wave in the tracings of healthy adolescents and young adults. Furthermore, the

fact that this one significant Q-3 wave led to the discovery that its possessor had frank valvular disease, and that his record had been improperly filed offers an illustration of the fact that this finding must not be passed over too readily. Pardee found two significant Q-3 waves in 227 records from presumably normal individuals. In both of these the position of the heart was more horizontal than the average.

The inclination of the heart in the body has been shown to influence the form of the electrocardiogram. In cases with high position of the diaphragm, significant Q-3 waves appear to be found more often than in controls. The disappearance of a significant Q-3 wave following delivery in two of our cases suggests that a change in position of the heart, as a result of an elevated diaphragm, had probably been responsible for the production of this wave. However, other conditions influence the heart during pregnancy and may possibly be factors in the production of this wave.

The percentage (6 per cent) of significant Q-3 waves found in 500 ward and clinic cardiovascular cases corresponds closely to the figure (7 per cent) obtained by Pardee in a study of 200 private cases. Willius,[5] however, found but 300 significant Q-3 waves in 70,000 electrocardiograms (0.04 per cent). The differences of incidence doubtless depend mainly on the type of material studied (Table III). Sixty-three

TABLE III

	TOTAL NUMBER WITH SIGNIFICANT Q_3 WAVES	ANGINAL SYNDROME	HYPERTENSION AND/OR ARTERIOSCLEROSIS WITHOUT THE ANGINAL SYNDROME	SYPHILITIC CARDIOVASCULAR DISEASE	RHEUMATIC TYPE HEART DISEASE
Pardee (number of cases not stated)	43	27 (63%)	2 (4.6%)	1 (2.32%)	4 (9.3%)
Edeiken and Wolferth; 500 ward and clinic cardiovascular cases	31	5 (16%)	10 (33%)	8 (25.8%)	3 (9.7%)
Willius, 70,000 electrocardiograms	300	115 (38.3%)	153 (51%)	7 (2.3%)	8 (2.7%)

per cent of the significant Q-3 waves in Pardee's group were found in patients suffering from the anginal syndrome, whereas the incidence was only 16 per cent in the 500 ward and clinic cases in this study. Willius found 38.3 per cent of all significant Q-3 waves in electrocardiograms of patients with the anginal syndrome. The percentage of deep Q-3 waves found in other heart conditions also varies in different studies (Table III).

In our series of cardiovascular cases, more Q-3 waves were discovered in patients with hypertensive cardiovascular disease (with no anginal

history) than in cases with the anginal syndrome (whether hypertensive or not). It is noteworthy that 8 of the 31 significant Q-3 waves in the group of 500 ward and clinic cardiovascular cases occurred in syphilitic cardiovascular disease. Although the number of cases is small, it would appear that the relationship cannot be regarded as accidental.

The incidence of Q-3 waves found in 1900 consecutive electrocardiograms (4.2 per cent) is, as might be expected, lower than that obtained in the five hundred ward and clinic cardiovascular cases (6 per cent).

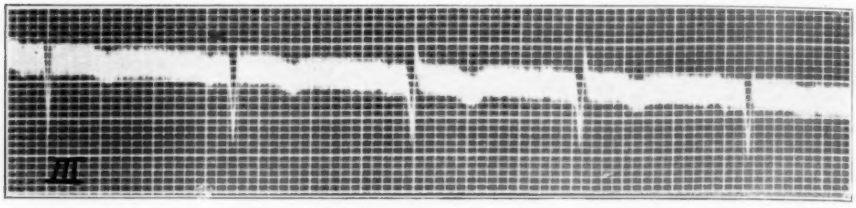

Fig. 2.—Variability of Q-3 wave in successive beats; respiration was not deep.

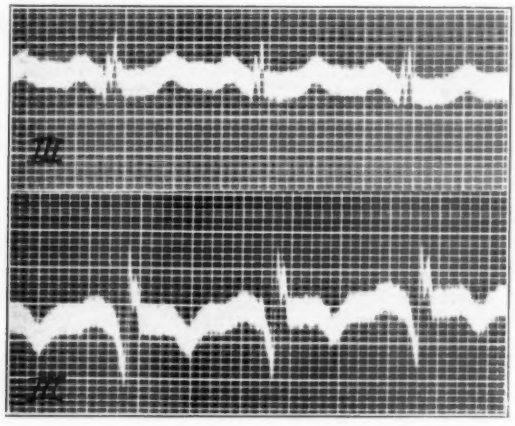

Fig. 3.—Lead III before and following coronary occlusion. Appearance of a significant Q-3 following occlusion. Interval between tracings was three and one-half months.

Among these 1900 there were many tracings made as part of a routine examination in patients who neither complained of cardiac symptoms nor showed evidence of cardiac disease on physical examination.

The high percentage (26.7 per cent) of significant Q-3 waves in the electrocardiograms of patients suffering from the anginal syndrome confirms the observation of Pardee that these patients show the deep Q-3 more often than cardiac patients in general. In Pardee's group of 200 unselected cardiacs, there were 30 cases of angina and 8, or 27 per cent, of these showed a significant Q-3. This incidence is almost identical with that of our group of 116 cases with the anginal syndrome.

Although a Q-3 wave was seen in 26.7 per cent of patients with the anginal syndrome and about 6 per cent of all cardiacs studied, the prob-

ability exists that it occurs in a much larger percentage of cases at one time or other. We have pointed out above that some of our cases of angina pectoris showed a significant Q-3 wave on one examination and failed to show one on another occasion (Fig. 1, *A* and *B*). As a matter of fact a single strip of tracing may show this variation (Fig. 2). Furthermore, the Q-wave may disappear during an attack of angina pectoris as shown in a record published by Wood, Wolferth and Livezey[6] (Fig. 5 of their paper).

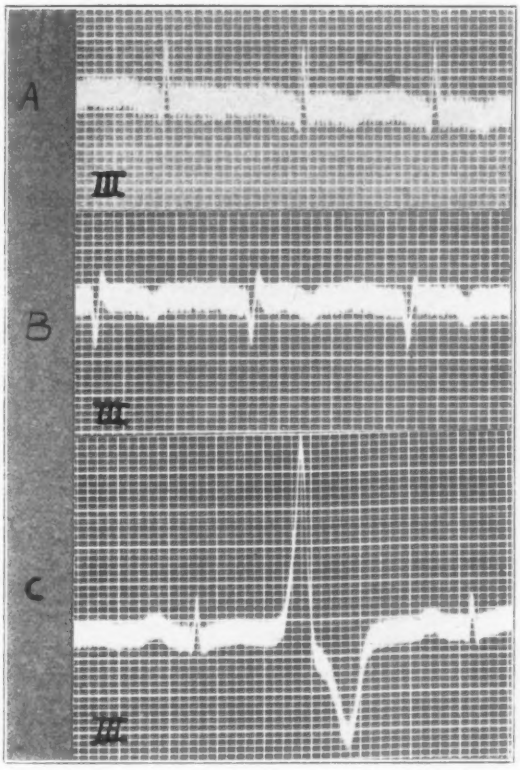

Fig. 4.—Lead III from patient with rheumatic heart disease. The three tracings were made over a period of one year; only one (*B*) showed a significant Q-3 wave.

Parkinson and Bedford[2] have noted the Q-wave in Lead III in some of their cases of coronary occlusion (9 times in 28 published cases). Fenichel and Kugell[7] have demonstrated the marked frequency of this wave in cases of occlusion with septal involvement. The possibility must be conceded, however, that the Q-wave might have been present in some of these cases prior to the occlusion. In Fig. 3 is shown one of three instances which we have seen of the development of a Q-3 wave (after a coronary occlusion) which had not been present prior to the occlusion.

The variability in the Q-3 wave is also observed in cases of rheumatic heart disease. In the electrocardiogram of a young man with rheumatic

mitral stenosis, during an attack of paroxysmal auricular tachycardia, a right axis deviation and a deep Q-3 wave were observed. Eight days later and five days after the cessation of the tachycardia under quinidine therapy, the right axis deviation and Q-3 were no longer present. In another case of rheumatic mitral stenosis (Fig. 4) only one of three tracings taken over a period of one year showed a deep Q-3 wave. In another case (Fig. 5) a significant Q-3 wave appeared only after an extrasystole.

The corporation executives used as a control, particularly for the anginal group, cannot be considered as an entirely normal group for some had demonstrable cardiac damage. The Q-3 wave was found in eight instances, and in seven of these there was definite evidence of

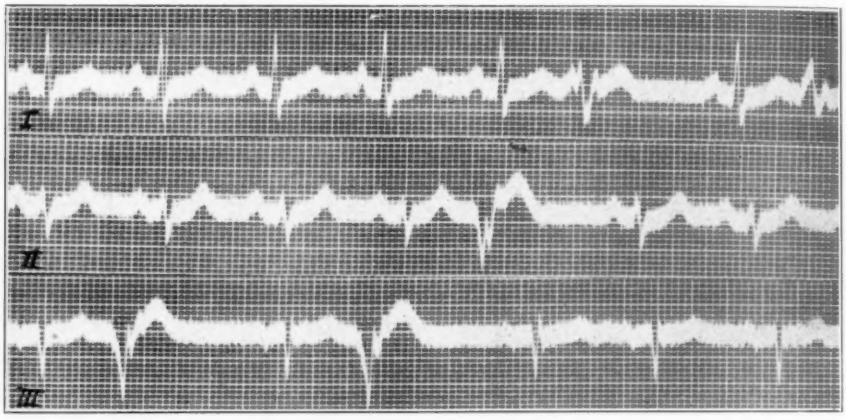

Fig. 5.—Electrocardiogram of a patient with rheumatic heart disease. A significant Q-3 wave appears after an extrasystole.

cardiac damage, although none complained of cardiovascular symptoms. Thus it would appear that the Q-3 wave is not only a fairly frequent finding in hypertensive or arteriosclerotic heart disease not sufficiently advanced to cause symptoms, but that when present it has considerable diagnostic value.

MECHANISM

It is generally believed that the interventricular septum is concerned in the production of the Q-wave. According to Lewis,[8] activation of the septum is responsible for the Q-wave and the beginning of the R-wave in all leads. It would seem, therefore, that increase in amplitude of the Q-wave beyond what is usually seen must be related to the spread of the excitatory process throughout the septum. On this basis, several possibilities that might account for a deep Q-3 wave suggest themselves: (1) The septum may be involved by a lesion which disturbs the spread of the excitatory process; (2) the septum may be increased in size; (3) the septum may be normal except for eccentricities in the distribution of the

conduction pathways; (4) the septum may be deviated from its usual position with reference to Lead III.

1. Fenichel and Kugell[7] found at necropsy a high incidence of involvement of the posterior part of the septum in the cases of coronary occlusion whose electrocardiograms exhibited a deep Q-3 wave. All of their 17 cases with large Q-3 revealed septal involvement at least in the posterior part. In the 16 cases without a large Q-3 only one showed involvement of the posterior portion of the septum. In material such as we report, however, it seems improbable that infarction of the septum, or other damage severe enough to modify the course of the excitatory process materially was present in more than a small minority of cases.

2. The suggestion that enlargement of the septum may be responsible for a deep Q-3 wave must remain for the present, at least, purely a speculation. The only point which we can make in support of this suggestion is the comparative frequency of deep Q-3 waves in subjects with hypertensive cardiovascular disease and enlarged hearts. On the other hand, the great majority of patients with marked enlargement of the heart and presumably some enlargement of the septum fail to show a deep Q-3 wave. Furthermore, many of the patients whose electrocardiograms exhibit a deep Q-3 wave, have no appreciable cardiac enlargement. Thus it seems unlikely that enlargement of the septum is a factor of major importance in accounting for the clinical incidence of the deep Q-3 wave.

3. Anomaly of pathways of conduction (aside from actual defect in conduction) is purely a theoretical conception that has been utilized previously, especially in the attempt to account for axis deviation in the absence of cardiac enlargement or displacement. Such a mechanism, as Pardee suggested, may be thought of as a possible explanation of the deep Q-3 wave in cases with normally placed hearts which show neither evidence of enlargement nor other pathological changes. The rarity of significant Q-3 waves in healthy young individuals, however, indicates that such an hypothesis has little if any practical application.

4. A factor which we believe deserves serious consideration, as a major cause of deep Q-3 waves, is change in position of the septum with reference to the plane of Lead III. There is considerable evidence to support such a view. The fact that on change in position one wave of the QRS complex may show marked changes without proportionate changes in other waves was demonstrated by Einthoven and deLint.[9] They showed that if an electrocardiogram was made with the subject lying on the left side and the procedure repeated with the subject on the right side, the S-wave, in Lead I of the second tracing tended to be deeper than in the first, although there was usually no material change in the R-wave.

In experiments on dogs, Meek and Wilson[10] demonstrated the marked influence of rotation of the heart on the QRS complexes of the electrocardiogram. According to these authors, either uncomplicated rotation of the apex to the right on the anteroposterior axis or rotation on the

longitudinal axis turning the front of the heart toward the left will give curves characteristic of right-sided preponderance. The opposite movements will give curves characteristic of left-sided preponderance. Mere displacement of the heart to the right or left usually causes a combined rotation on both anteroposterior and longitudinal axes and produces electrocardiographic changes which cannot be predicted because of the opposing effects of simultaneous rotation around the anteroposterior and longitudinal axis. For example, pulling the heart to the left produced left axis deviation in the majority of cases but also caused an increased incidence of Q-3 from 45 to 68 per cent in 30 experiments. Q-3 is frequently seen in electrocardiograms with right axis deviation and is seldom found in left axis deviation. Pulling the heart to the right side did not produce a right axis deviation; Q-1 either appeared or, if already present, increased in 26 of 29 cases (90 per cent) and Q-3 was found in

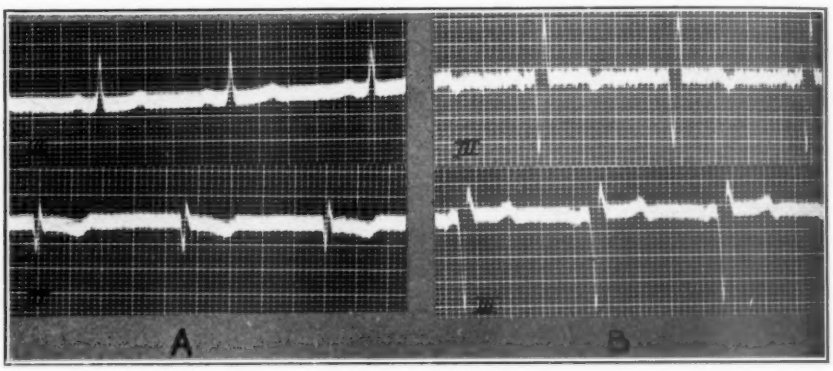

Fig. 6.—*A*, Production of a deep Q-wave by transferring the left leg electrode to the region of angle of left scapula (lower tracing). Upper tracing shows Lead III taken in conventional manner. *B*, A significant Q-3 wave made deeper by the above procedure.

only 34 per cent of all records, a decrease of 11 per cent from normal. This is not what was expected, since Q-1 is seldom found in right axis deviation. However, by limiting the movement of the heart to rotation around its longitudinal axis or by pulling the heart to the left or right and then correcting for the longitudinal rotation, they could produce tracings typical of left and right axis deviation.

The observations of Meek and Wilson, therefore, have a bearing on (a) the varied effects on the electrocardiogram produced by attempts at rotation of the heart in the human, and (b) the findings of a deep Q-3 wave in electrocardiograms with left axis deviation. We attempted to produce a significant Q-3 wave in subjects with presumably normal hearts by taking tracings with the body in various positions. Although marked changes in QRS complexes were produced by changes in position of the body and presumably of the heart, Q-3 was not produced. However, by rotating the plane of the lead in relation to the heart, the electrodes can

be so placed that a Q-wave is produced. This can be done by leaving the left arm electrode in position and transferring the left leg electrode to the region of the angle of the left scapula (Fig. 6, *A*). By the same type of procedure a Q-wave already present can sometimes be made proportionately deeper (Fig. 6, *B*). In certain cases, however, Lead III taken with the patient in the upright position may be the most favorable procedure for recording the Q-wave. Thus in one patient with aortic insufficiency and mitral stenosis, the electrocardiogram with the patient in

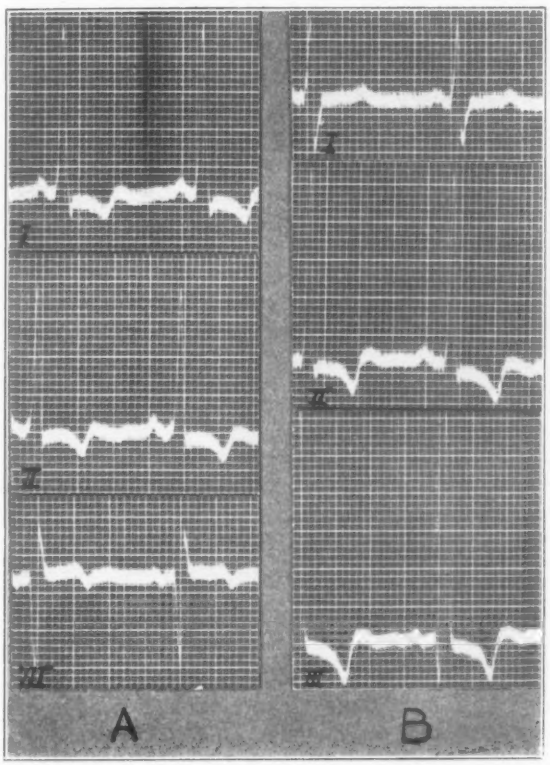

Fig. 7.—Records showing effect of position of body upon a deep Q-3 wave. *A*, Patient in sitting position. *B*, Tracing taken immediately after *A*, with patient on left side.

the upright position showed left axis deviation and a deep Q-3. By changing the position of the patient, it was possible to produce right axis deviation and at the same time diminution of the Q-3 wave (Fig. 7).

From the above considerations, it would appear that not only may rotation of the septum determine the presence or absence of a deep Q-3 wave but the degree of rotation need not be great. The factors which determine such rotation in human hearts are not well understood and must be studied further if the mechanism of the deep Q-3 wave is to be worked out. For the present we must content ourselves by calling at-

tention to the importance of the position of the septum with reference to the plane of the electrocardiographic lead. The reasons why a deep Q-3 wave in association with a normal electrical axis or deviation to the left occurs in a certain proportion of cases with evidences of myocardial damage and is rare in cases with presumably healthy hearts remain obscure.

SUMMARY

1. In the electrocardiograms of 709 apparently normal college students, no significant Q-3 waves were discovered. Among 117 college athletes, the only significant Q-3 wave occurred in an individual with rheumatic heart disease.

2. A significant Q-3 wave was found in approximately 6 per cent of 500 hospital, ward and clinic patients with cardiovascular disease. Although all ages were included, the youngest patient to show a significant Q-3 wave was thirty-two years of age; the oldest was seventy-two years. Hypertension, arteriosclerosis and syphilis were regarded as the etiological factors of the heart disease in the majority of cases, but significant Q-3 waves were also seen in cases of rheumatic and thyrotoxic heart disease.

3. In nineteen hundred unselected electrocardiograms taken from the files of the Cardiographic Laboratory, including ward, clinic and private patients of all ages, there were 78 (4.1 per cent) significant Q-3 waves. Of the 78 individuals exhibiting the significant Q-3 waves, 63 were classified as having heart disease, 7 were placed in a doubtful group, 5 were considered negative, and 3 were not classified because of inadequate records.

4. Thirty-one or 26.7 per cent of one hundred and sixteen cases of the anginal syndrome showed a significant Q-3 wave.

5. Eight or 5.5 per cent of the electrocardiograms of one hundred and forty-five corporation executives showed a significant Q-3 wave. In seven of these eight cases there was definite evidence of cardiovascular disease, although the history in all was negative.

6. A total of 149 significant Q-3 waves was found in the first five groups of this study. The electrocardiogram was otherwise normal in 68 cases; 64 showed T-wave changes; and 17 other electrocardiographic abnormalities. This sign, therefore, may constitute the first electrocardiographic change to invite attention to the possibility of cardiac damage.

7. Twenty-five pregnant women in the ninth month, were electrocardiographed, and three were found to show significant Q-3 waves. In two of these who were reexamined, several months after delivery, the Q-3 wave had vanished.

8. Available evidence indicates that the conditions chiefly responsible for deep Q-3 waves are either (1) a lesion of the septum which interferes with the spread of the excitatory process, or (2) deviation of the septum

from its usual position with reference to Lead III of the electrocardiogram.

9. Study of the clinical incidence of the deep Q-3 wave suggests that in the majority of cases change of position rather than injury severe enough to alter the spread of the excitatory process, is the important factor in the production of this wave.

10. Deep Q-3 waves which conform to the criteria of Pardee are occasionally present in electrocardiograms of patients who show neither evidence of cardiac disease nor changes from the normal in the size and position of the heart. Whether or not this is due to some peculiarity in the ventricular tissues first invaded by the excitatory process, cannot be stated. It is important, however, to take these cases into account when the attempt is made to evaluate the clinical significance of the deep Q-3 wave.

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HEART DISEASE IN THE AMERICAN NEGRO OF THE SOUTH*

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A RECENTLY reported investigation[1] disclosing the greater prevalence of organic heart disease in the negro as compared with the white race makes pertinent an examination into the incidence and course of this group of diseases in the black race, a subject heretofore inadequately considered.[2, 3, 4]

Among 10,188 new patients (4,252 white and 5,936 negro patients) seen in the Medical Division of the Dispensary of the John Sealy Hospital during the past seven and one-half years, there were found 1,660 cases of organic heart disease. This group comprised 488 white patients and 1,172 negro patients, which makes the incidence of organic heart disease in medical patients for the two races 11.5 and 19.7 per cent, respectively. It follows from this analysis that in the medical patients seen in this Dispensary organic heart disease of all varieties occurs one and seven-tenths times more often in the negro than in the white race.

The average age of the negroes with all types of organic heart disease was found to be 46.6 years, whereas the average age of the white patients in the series was 56.3 years. Manifestly, organic heart disease of all varieties occurs on the average 9.7 years earlier in the life of the negro than in the white patient.

That the mortality rate from heart disease in the negro is higher than that in the white race is a well established fact. Dublin,[5] in studying the death rates from the Industrial Department of the Metropolitan Life Insurance Company, found that negro males show death rates from heart disease, during the main period of life, from 65 to 80 per cent higher than white males of the same ages and for negro women the rates are twice as high as for white women of similar age periods. Corroborative of these facts is the report of Woody[6] who, in a study of death rate statistics in the southern states, arrived at similar conclusions. Holt[7] compared the death rates of the white and negro races in Little Rock and Arkansas and found the death rate from heart disease to be 80 per cent higher in the negro than in the white race.

Not only is the mortality rate from heart disease higher in the negro than in the white race, but, likewise, the resulting morbidity is of much greater degree. It has long been a common observation of those who see many negro patients that a diagnosis of organic heart disease in the

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case of a negro is of much more serious import than a similar diagnosis given a white patient. Such, it is offered, would be anticipated in view of the fact that the economic status and mode of living of the negro would prevent him from caring for himself in a manner that the white patient could and would. However, it should be added that the same difficulties are encountered in the treatment of heart disease in any individual, regardless of race, who is dependent upon manual labor for sustenance. The majority of the white patients with whom we have compared the negro in this regard were of practically the same social stratum and worked and lived under similar conditions. Therefore, logical as the explanation cited may at first appear, it fails to reconcile the discrepancy in the mortality and morbidity rates from heart disease in the two races.

HYPERTENSIVE CARDIOVASCULAR DISEASE

The prominence of syphilis as a cause of heart disease in the negro is universally appreciated; however, the rôle played by hypertension as a causal agent of heart disease in this race has failed to receive the emphasis which its importance as such a factor warrants.

In our initial investigation previously referred to, it was found that the incidence of hypertensive cardiovascular disease in the patients coming to the Medical Division of the Dispensary was 12.5 per cent for negroes and 4.9 per cent for white patients from which it follows that this type of heart disease in medical patients in this Dispensary is of two and one-half times greater incidence in the negro than in the white race. Analysis of these same data in regard to both race and sex revealed the following incidence figures: negro males 10.6 per cent, white males 3.7 per cent, negro females 14.7 per cent, and white females 6.6 per cent. These figures justify the conclusion that in this clinic this type of heart disease is of three and one-tenth times greater incidence in the negro male than in the white male, and of two and two-tenths times greater incidence in the negro female than in the white female. It is also obvious that hypertensive cardiovascular disease in this clinic occurs one and one-half times more often in the negro female than in the negro male.

A comparison of the age incidence in the negro and in the white patients with this form of heart disease is in point, and the discrepancy in this regard is sufficiently striking to merit a detailed consideration. The average ages for patients with hypertensive heart disease were found to be as follows: white males 54.6 years, white females 51.1 years, negro males 47.7 years, and negro females 43.4 years. The conclusion is therefore justified that in this clinic hypertensive heart disease occurs, on the average, seven years earlier in the life of the negro than in that of the white patient. An analysis of the ages of the patients in regard to the occurrence of hypertension by decades revealed that in white

patients the cases were distributed as follows: 20 to 30 years, 0.5 per cent; 30 to 40 years, 3.4 per cent; 40 to 50 years 30 per cent; 50 to 60 years, 40.4 per cent; 60 to 70 years, 23.1 per cent; 70 to 80 years, 2.5 per cent. These figures are in considerable contrast to those for the negro race which were as follows: 10 to 20 years, 0.13 per cent; 20 to 30 years, 5.5 per cent; 30 to 40 years 25.1 per cent; 40 to 50 years, 34.2 per cent; 50 to 60 years, 26.1 per cent; 60 to 70 years, 8.2 per cent; 70

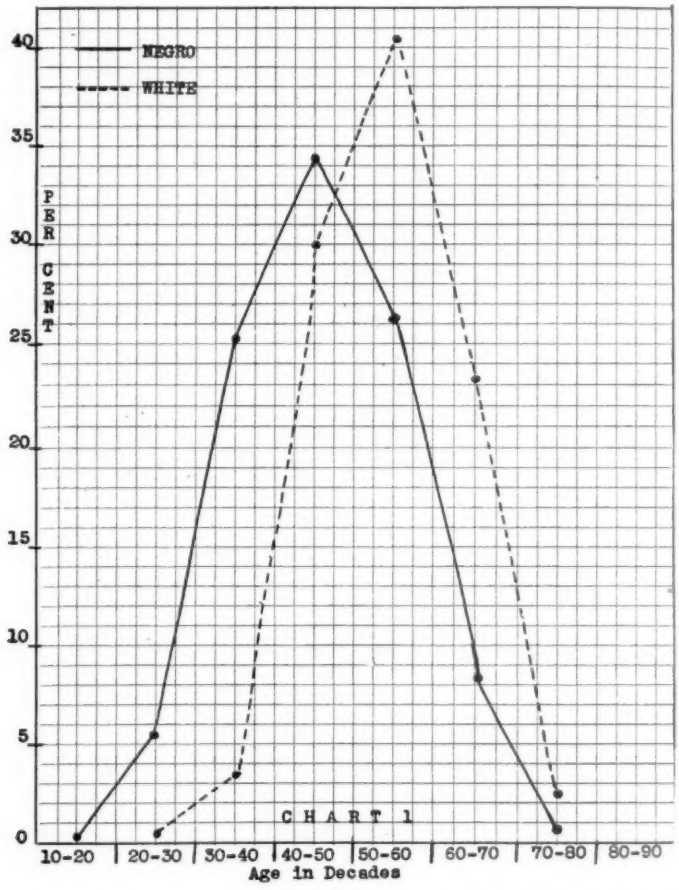

Chart 1.—A comparison of the occurrence by decades of hypertensive cardiovascular disease in the white and negro races.

to 80 years, 0.7 per cent. Thus, it is seen that the greatest incidence of hypertensive heart disease in the negro race occurs in the fourth decade, whereas in the white race the peak is reached in the fifth decade. And it is likewise apparent that 65 per cent of the cases of this type of heart disease in the negro occur prior to the fiftieth year of life, whereas in the white race 65 per cent of the patients had already passed that period in life. The most marked difference in incidence lies in the third

decade at which time vascular hypertension is of nearly eight times greater incidence in the negro than in the white race. The differences in the two races in this regard are depicted graphically in Chart 1. A comparison of data computed in a like manner with reference to the occurrence of hypertension in negro males and females reveals that this condition occurs earlier in the life of the female than in the male. This compilation discloses that approximately 71 per cent of the cases of

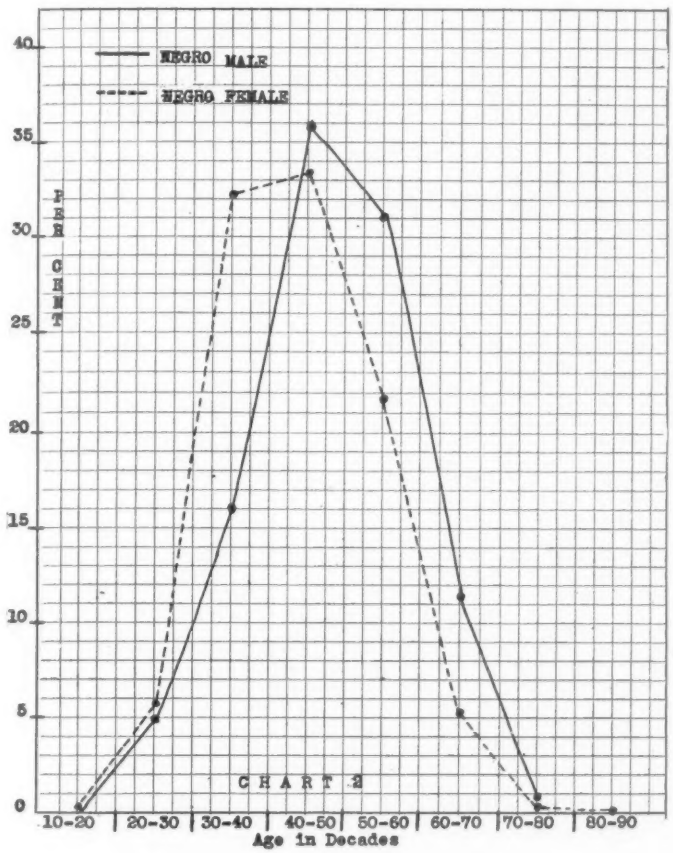

Chart 2.—A comparison of the occurrence by decades of hypertensive cardiovascular disease in negro males and females.

hypertension in the negro female occur prior to the fiftieth year whereas only 57 per cent of the cases in the negro male appear prior to that period in life. Likewise, it is manifest that the incidence in the third decade is twice as great in the female as in the male negro (Chart 2).

Just as there is considerable difference in the two races in regard to the incidence and age of occurrence, so the course of hypertensive cardiovascular disease in the negro is quite unlike that in his white brother. Although our patients were not subclassified into benign, severe benign,

and malignant types as suggested by Keith and his associates,[8] a small experience with negroes who have hypertensive cardiovascular disease is sufficient to impress one with the logic and merits of the use of the term "malignant hypertension," for in the negro and particularly the negro in the third decade of life, the hypertension is frequently of a most fulminating type, death occurring early from what often appears to be a combined insufficiency of the heart, kidneys, and brain. The course of the disease in general is much more rapid, the complications promptly follow recognition of the hypertension, and death occurs much sooner than would ordinarily be anticipated in the white patient with this disease. The inevitable complications of essential hypertension manifest themselves in the negro predominately in the heart and kidneys. Cerebral hemorrhage is a much less frequent cause of death in the negro than in the white race. A study of the causes of death obtained from postmortem examinations in a small series of negroes dying from hypertension and its complications yields results appreciably different from those reported by Bell and Clawson[9] whose series, we assume, includes only white patients. Considerable difficulty was encountered in many instances in determining the true cause of death, as in many cases renal and cardiac factors were inseparable. It should be mentioned that many of the deaths due to uremia were preceded by one or more breaks in compensation. Although the study is not yet complete, it appears that the incidence of myocardial deaths was about the same, renal deaths were about twice as common in the negro, and cerebral deaths, hemorrhage and encephelomalacia, only about one-half as frequent in the negro as in the white race.

SYPHILITIC CARDIOVASCULAR DISEASE

In the investigation referred to above,[1] heart disease of this type was found to occur four times more often in the negro than in the white patient, the percentages for the respective races being 3 per cent for the former and 0.7 per cent for the latter. The average age of the negro patient with this type of cardiovascular disability was 40.6 years and for the white patient 45.5 years.

That syphilis is much more prevalent in the negro than in the white race remains an undisputed fact. Nevertheless, we do not believe that the discrepancy in the incidence of syphilis in the two races is sufficient to account for the fact that syphilitic heart disease occurs four times more frequently in the negro race, more particularly when one considers that the members of the two races in this series were essentially of the same social stratum and economic status. Furthermore, in a given number of patients with syphilis equally divided in number as to race, it is our belief that a larger number of negro patients will develop cardiovascular syphilis than will white patients. Stone and Vanzant[2] have offered the opinion that the cardiovascular apparatus of

the negro is more susceptible to syphilitic infection than that of his white brother. The appearance of cardiovascular syphilis at an earlier age in the negro can be explained in part by the earlier sexual maturity of the negro who, consequently, is exposed to venereal infection earlier in life. It is probable that hard manual labor, though it does not enhance the predilection of the spirochete for the cardiovascular system, may be a factor in aggravating the resulting morbid physiology with consequent earlier symptomatic manifestations.

The relative paucity of cases of uncomplicated syphilitic aortitis (14) compared with the number of cases of aortic regurgitation (134) and aneurysm (31) appearing in the series referred to merits consideration. We are convinced that the characteristic clinical picture of syphilitic aortitis with substernal pain and nocturnal attacks of dyspnea occurs very rarely in the negro. Of all patients placed in this category none had classical symptoms, the diagnosis being made from physical findings, roentgenographic, and serological studies.

The statements made concerning the course of heart disease in general in the negro race are particularly true in the case of cardiovascular syphilis. Heart failure of this etiology is the beginning of an end near at hand. The few who survive the first failure make a partial recovery and live the life of cardiac invalids for a few months at the most.

ARTERIOSCLEROTIC HEART DISEASE

The remaining important etiological group in this locality, namely, the arteriosclerotic or coronary artery disease group, differs from the two types previously considered in that it was found to be more prevalent in the white race. The incidence for this type in the medical cases reviewed was 2.7 per cent for the negro and 4.1 per cent for white patients. From this analysis the conclusion is permissible that this type of heart disease in this clinic is of one and one-half times greater incidence in the latter than in the former race. It should be remembered in this connection that arterial disease in advanced stages is by no means uncommon in the American negro. The discrepancy in the incidence of this etiological type in the two races is probably accounted for in a large measure by the fact that a greater number of the white race reach that period in life in which degeneration of the vascular tree comomonly occurs. This explanation is corroborated by the findings of Holt[7] who showed that the average duration of life in the negro was 8.3 years less than that in the white race. The opinion is offered that in those negroes reaching the sixth and seventh decades, the incidence of the arteriosclerotic type of heart disease would be greater than that in the white race. This type of heart disease was found to occur, on the average, nine years earlier in the life of the negro than in the white patient. This fact is perhaps very significant in that it strongly suggests an inherent inferiority of the cardiovascular system of the negro.

ANGINA PECTORIS

In the 10,188 medical cases studied, only eleven cases of angina pectoris were found. The low incidence for this community is in accord with the findings of other observers[2, 4] in the South, and is in contrast to the observations made by investigators[10, 11] in the North and East where angina pectoris appears to be much more common. A more relevant revelation is the fact that all of the 11 cases occurred in white patients; in the 5,936 negro medical patients examined, the syndrome of angina pectoris failed to be discerned in a single instance. This discrepancy in the occurrence of angina pectoris in the two races is difficult to explain, particularly because of the inadequacy of our knowledge concerning the pathogenesis of this symptom complex. We can be almost certain that the cause for this difference does not lie in a dissimilarity of the pathological and functional changes to which the cardiovascular systems of the two races are subjected for the reason that these morbid processes are seen to occur more frequently and to be of a severer degree in the negro than in the white race. The possibility of there being an inherent anatomical difference in the innervation of the heart and great vessels in the two races is so remote that it commands no consideration. Roberts[12] feels that the absence of angina pectoris in the negro is to be accounted for by the fact that nervous and mental strain does not enter into the life of the negro, whereas it is a potent factor in the white races of Western Europe and North America. Certainly, there is a profound dissimilarity in the psyche and sensorium in the two races under consideration. Therefore, it seems logical to assume that the basis for the discrepancy in the occurrence of this syndrome lies in an inherent difference in the sensitivity of the nervous systems in the two races.

OTHER TYPES OF HEART DISEASE

Unfortunately, the paucity of material representative of the rheumatic and thyrotoxic types of heart disease in the series studied does not permit consideration sufficiently satisfactory to merit the drawing of conclusions. The incidence of these types of heart disease in the group investigated was practically the same for the two races despite the fact that rheumatic fever and hyperthyroidism are appreciably more prevalent in the white race.

SUMMARY

In view of the facts that heart disease in the negro as compared with the white race is of greater incidence, occurs at a younger age, pursues a more rapid course, and has a higher mortality rate, the opinion is offered that the cardiovascular system of the American negro of the South is inferior to that of the white race, and is more vulnerable to insult whether this be applied as an infection, a degeneration, a toxemia, or in

the form of the stress and strain incident to the complexities and modes of modern occidental civilization.

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THE ELECTROCARDIOGRAM IN LATE MIDDLE LIFE*

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INTRODUCTION

ELECTROCARDIOGRAPHIC diagnosis is increasingly concerned with the early recognition of degenerative heart disease occurring in late middle life, and minor changes are often emphasized as indicative of early myocardial damage. Since ordinarily the tests are made because of complaints referable to the cardiovascular system, curves representing the normal heart for this age of life have not been extensively studied. Such series of healthy young adults as have been obtained by Lewis and Gilder,[1] Cohn,[2] and Ferguson and O'Connell,[3] have not dealt with the normal heart in persons over fifty years of age. The purpose of this study was to contribute to the recognition of the normal electrocardiogram for this age and to determine if there are in elderly persons any changes which, though not common in the normal tracings of younger people, must nevertheless be considered physiologic at this stage of life.

PROCEDURE

Fifty persons between the ages of fifty and sixty-five were selected. They had been admitted to the Barnes Hospital or the Washington University Clinics for a variety of complaints none of which were thought to implicate the cardiovascular system. The patients were excluded if they suffered from marked debility or from an illness which was considered very serious. All of the patients were carefully questioned and denied the presence of symptoms of cardiac disease. They denied all history of heart trouble or of rheumatic fever. On physical examination there were no abnormal findings in the heart. The blood vessels were considered normal for their ages. Specifically, there were no murmurs, demonstrable enlargement of the heart, cardiac irregularities except occasional extrasystoles, and no blood pressures were found to exceed 150 mm. systolic or 90 diastolic. In 43 cases six-foot x-ray plates were taken and the heart measurements were within normal limits. The Wassermann reaction was negative in all cases.

The electrocardiograms were taken with the patients in two positions, sitting (Position 1) and supine (Position 2). A Hindle machine was used in both positions and in addition, for a comparison between the two technics, a Victor machine was employed for the supine position only.

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FINDINGS

P-Waves.—In Lead I the P-waves were upright and normal in 36 cases. In 10 cases they were indeterminate while they were upright in the other leads; in 3 cases they were indeterminate in Leads I and III. They were, in one case, indeterminate in Leads I and II. In no case were they inverted, so it was concluded that the P-waves in Lead I showed very little variation from normal, and then merely a tendency to become iso-electric.

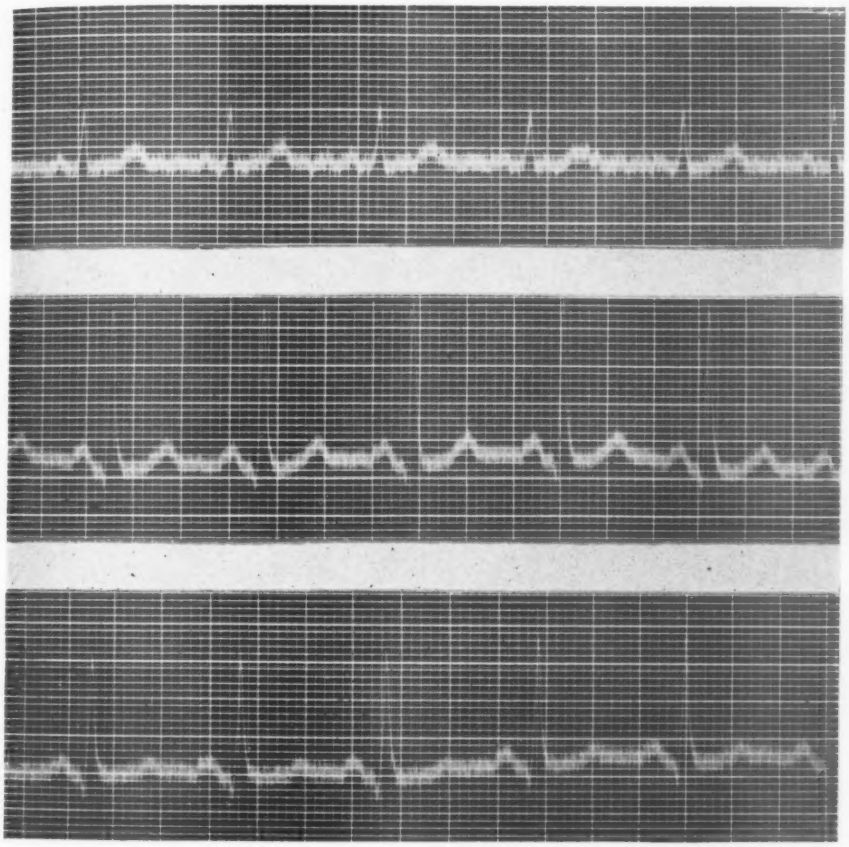

Fig. 1.—Case No. 58, Position 1, showing definite Q-waves in Leads II and III.

In Lead II the P-waves were uniformly high and normal in 49 cases. In 3 of these they changed with position, being higher in Position 1. They were almost iso-electric in one case and had a downward phase in one case. It was concluded that the P-wave in Lead II showed remarkably little variation from the accepted normal and then merely a change with change in position.

In Lead III the P-waves were normal and upright and did not change with position in 31 cases. They were indeterminate in 13 cases; in one

of these they were indeterminate in Position 1, but upright in Position 2, in 2 they were indeterminate in Position 2, but upright in Position 1. The P-waves were inverted in 4 cases and in all of these they became upright on deep inspiration. In one case the P-waves were higher in Position 2 than in Position 1 and in one case higher in Position 1 than in Position 2.

Considering all three leads together it has been found that in 17 cases the P-waves were upright, normal in shape, and not affected by position or respiration. Altogether they showed no difference from what may be expected at any age.

QRS Complexes.—In Lead I the complexes consisted of an R-wave only in 21 cases; in 7 there was a definite Q-wave; in 2 of these it was associated with slurring of the downstroke of the R-wave; in one the

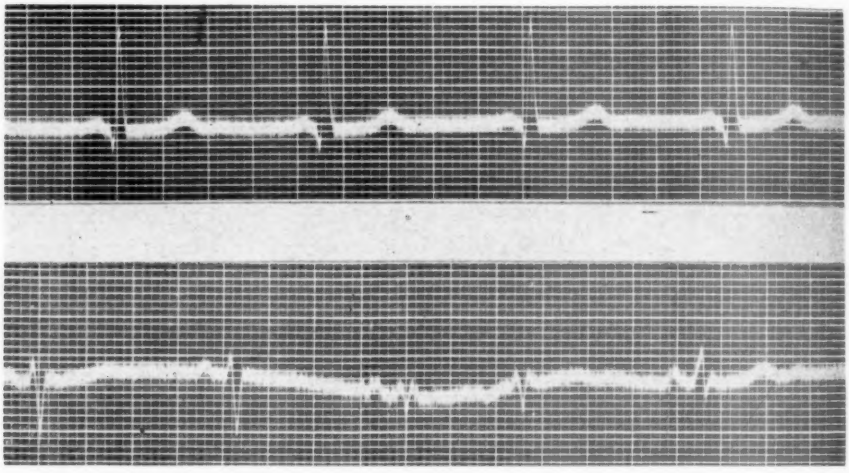

Fig. 2.—Case No. 7, Position 1, Leads I and III showing definite Q-waves in Lead I and transverse heart in Lead III.

R-wave was unusually high and sharp and in one both Q-wave and S-wave were definite.

In 4 cases the S-wave was very deep in both positions; in 3 other cases it was marked in one position only, in 2 cases more marked in Position 1 than in Position 2, in 1 more marked in Position 2 than in Position 1. In 1 case only was it sufficiently marked to constitute right axis deviation.

Slurring of the upstroke of the R-wave occurred in 2 cases, and of the downstroke in 8 cases; in 1 of these it was interpreted as high branching. In 5 cases there was slight slurring of the entire complexes. In one case the voltage was very high and in another very low.

In Lead II the Q-waves were marked in 3 cases, in 1 case more in Position 1 than in Position 2. In 1 case the Q-wave was accompanied by a slurring of the descending R-wave.

The descending limb of the R-wave was slurred in 6 cases; 2 of these

also showed "high branching"; 1 showed a notch like an S-wave during the downstroke. Slurring of the ascending R occurred in 3 cases, in 1 both upstrokes and downstrokes were slurred. In 13 cases there was an R-wave only.

The S-wave was marked in 18 cases, in 1 more so in Position 1 and in 2 more so in Position 2. In 3 of the 18 cases the S-waves were both wide and slurred.

The entire QRS complex in Lead II was slurred in 5 cases; in 1 of these the slurring was more marked in Position 1. In 2 cases the complexes were low, in 1 of these it was more pronounced in Position 2.

In Lead III the Q-wave was absent in 35 cases, its presence was doubtful in 5 cases, in 10 it was present and well defined, in 4 of these it exceeded 25 per cent of the greatest excursion of the QRS complex, thus

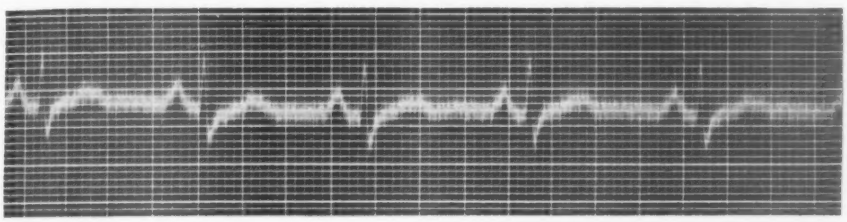

Fig. 3.—Case 43, Position 1, Lead II showing marked slurring and widening of S-wave.

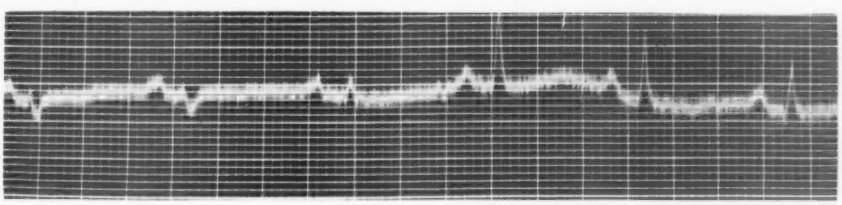

Fig. 4.—Case 45, Position 1, Lead III showing small slurred complexes becoming sharp and upright on deep inspiration.

complying with Pardee's criterion.[4] In 3 cases there was an inverted complex and it could not be determined with certainty whether this was a Q, an S, or an inverted R-wave. Change in position caused the Q-wave to change in 7 cases, in 2 it was deeper in Position 2 than in Position 1, in 5 it was more marked in Position 1 than in Position 2.

The R-wave was slurred in 48 cases and in 35 of these it was also low, in the other 13 it was of moderate height. In 16 cases the R-wave became markedly higher on inspiration; in 1 case inspiration resulted in notching of the R-wave. In 5 cases the R-wave was notched; in 5 cases the height of the R-wave changed with change in position.

The S-wave was present and distinct in 29 cases, in 14 of these it was somewhat slurred. In 15 cases it changed with change in position; in 12, it was greater in Position 1 than in Position 2, and in 3 it was greater in Position 2 than in Position 1. In addition to the 4 cases of transverse

heart as evidenced by inverted Q-waves there were 7 cases of transverse heart in which the inverted wave was clearly an S-wave. In 7 cases the inversion was deep enough to be called left axis deviation. Deep inspiration affected the wave in 14 cases, in 11 cases it became shallower, in 3 cases it became deeper. In 1 case it apparently changed from a negative to a positive wave on deep inspiration. S-waves were absent from Lead III in 21 cases.

T-Waves.—In Leads I and II no T-waves were inverted. In 2 cases the T-waves were iso-electric in Lead I, Position 1; in both of these they were upright in Position 2. In 1 case they were diphasic in Lead II, Position 1, but became entirely upright in Position 2 (in this case the T-wave was inverted in Lead III, more in Position 1 than in Position 2).

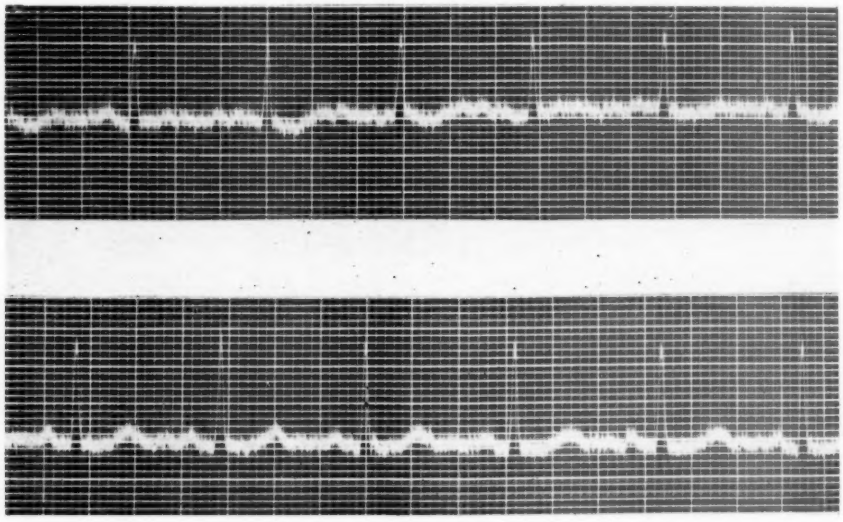

Fig. 5.—Case 6, Lead II, Positions 1 and 2, showing change of T-waves with change of position.

In 15 cases the T-waves were upright in all leads. In 2 of these the T-waves in Leads II and III were greater in Position 2 than in Position 1. In 25 cases the T-waves were upright in Leads I and II; in 14 of these they were iso-electric in Lead III, in 5 they were diphasic in Lead III, and in 6, inverted in Lead III; of these 6, 3 were more inverted in Position 1 than in Position 2.

Change in position affected the T-waves: in Lead II they were in 3 cases higher in Position 2 than in Position 1; in 1 of these the T-wave in Lead III was indeterminate in Position 1 while it became upright in Position 2, in another case the T-wave in Lead III was diphasic in Position 1 and became upright in Position 2.

In general, it was concluded that the T-waves showed very few changes in Leads I and II, but that in about half the cases the T-waves showed in Lead III changes from the simple upright shape.

Measurements.—Three time intervals were measured; the P-R interval, the duration of the QRS complex, and the S-T interval. All measurements fell within normal limits.

Of the P-R intervals 43 were between 0.15 and 0.19 seconds; 3 were 0.20, and 4 were 0.12 to 0.14.

Of the QRS complexes 43 were between 0.06 and 0.08 seconds, 3 were 0.09, and four were 0.04 to 0.05 seconds.

Of the S-T intervals, 47 were between 0.26 and 0.32 seconds, the other 3 readings were 0.24, 0.34, and 0.36 seconds.

There was thus no tendency to prolongation of any intervals, especially not to spreading of the QRS complex.

DISCUSSION

The question may be raised whether all of the patients in this series really had undamaged hearts. If it is true, as Fahr[5] states, that 23 per cent of deaths among persons past fifty years of age are caused by heart disease it is not unlikely that some persons in this series had myocardial disease which had not yet caused symptoms or physical findings. Then among, say, any 60 persons past fifty years of age, 14 may statistically be expected to die from heart disease. But if we exclude from the 60 those among the 14 who have manifest symptoms of heart disease or abnormal findings, such as increased blood pressure, cardiac enlargement, murmurs or cardiac irregularities, and those who have not yet developed any pathological process, and who, therefore, must be considered healthy, the chances are, that after such analysis we shall be left with a series of about 50 persons among whom very few have "hidden" heart disease. We believe that the electrocardiograms just described are from such a series. Therefore, in the absence of any changes which we know by experience are associated with grave cardiac disorders, the findings of this series may probably be considered such as occur in normal persons. On the other hand, the absence of certain findings from the series should cause these to be looked upon with suspicion as possibly indicating cardiac pathology.

SUMMARY

Within the limits of our experience we may state:

The following findings, some of which we have previously considered of doubtful significance, are not pathological.

Diphasic or iso-electric P-waves in Lead I or inverted P-waves in Lead III, if they become upright on deep inspiration; slurring of QRS complexes, especially in Lead III and slight to moderate notching of R with the QRS interval below 0.10 seconds, "transverse heart," isolated left axis deviation, moderate inversion of T-III (this last finding is very common).

On the other hand, the following findings were not present in this series and must therefore be looked upon with suspicion.

Indeterminate or inverted P-waves in Leads I or II, inverted P-waves in Lead III, if they do not become upright on deep inspiration. Inversion of T-waves in Leads I or I and II, or iso-electric T-waves in Leads I or II if they do not become upright on deep inspiration. A P-R interval exceeding 0.20 seconds, a QRS interval exceeding 0.10 seconds, or an S-T interval exceeding 0.34 or 0.36 seconds (the upper limit seems slightly uncertain).

It is seen that for the proper evaluation of a doubtful finding deep inspiration and sometimes change in position may be necessary in any lead. Such thorough investigation should always be done when the electrocardiographic findings may be the determining factor in the diagnosis.

This material does not indicate whether an isolated right axis deviation is definitely a pathological sign or whether the inverted wave in a transverse heart is always an S-wave.

There do not seem to be any characteristic "age-changes" in the electrocardiogram.

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VENTRICULAR TACHYCARDIA: AN INTERPRETATION OF THE NATURE OF ITS MECHANISM*

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THE stroke volume output of the mammalian ventricle is largely dependent upon a uniform and nearly simultaneous contraction of all parts of its musculature. This is brought about by the highly specialized His-Tawara-Purkinje system with its specific anatomical arrangement and conduction capacities. From the A-V node the excitation proceeds by the bundle branches to both chambers and spreads through the rapidly conducting network of Purkinje to all parts of the ventricles. This plan of transmission is in contrast to that in the auricles where the excitation is distributed by direct propagation through the more primitive arrangement of the muscle bands.

Tachycardia, flutter, and fibrillation of auricular origin are considerably more common than are the similar phenomena in the ventricles. In a previous study of ventricular fibrillation[1] it was suggested that the probable explanation of this discrepancy lies in the normal preventive and regulatory action effected through the His-Tawara-Purkinje system; that its capacity to distribute supraventricular impulses very quickly to all regions of the musculature not only insures harmony of contraction but further creates a uniform state of refractoriness which in itself tends to resist the setting up of circus movements in these chambers. An ectopic impulse tending to perpetuate itself through a process of reexcitation would theoretically be frustrated by a supraventricular impulse which at the opportune moment spreads through the normal path to all parts of the ventricle and induces a state of refractoriness sufficient to block the path of the re-entrant excitation. This would account for the rarity of continuous runs of ectopic beats in healthy individuals, for isolated premature beats are common, and in psychoneurotic individuals, a bigeminal rhythm is not infrequent. If the presence of frequent premature beats may be taken as an expression of hyperirritability of the ventricles, an explanation of the occurrence of ventricular tachycardia based on this factor alone would appear inadequate.

In bundle-branch block the ventricle corresponding to the side of disease receives its excitation from the adjacent ventricle by direct propagation through the septum. The spread is similar to that present in the ventricular premature beat. So long as conduction is still satisfactory through one bundle branch, the rhythm may continue to be governed through the A-V node. When conduction, however, is seriously impaired in both bundle branches, conditions are opportune for the development of circus movements, or tachycardias. The coordinating and regulatory function of the His-Tawara-Purkinje system is lost;

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the path through which impulses normally interrupt a sequence of ectopic beats is impaired; and the ventricles are exposed to any aberrant excitation. In the event of complete failure of nodal activity, and in the absence of a spontaneous excitation in the musculature, ventricular standstill ensues.

Explanations for the development of ventricular tachycardia have generally failed to give full consideration to the state of the conducting tissues and have invariably emphasized the condition of the myocardium, a hyperirritability induced by underlying disease, digitalis, or a combination of both. In the present communication attention is called to the fact that disorders of the ventricular rhythm such as tachycardia or flutter fibrillation are in many instances intimately related to direct interference with the His-Tawara-Purkinje system of conduction, and that disease of the bundle tissues, or depression of these tissues by drugs is an important precursory state in their development. The same interpretation as related to ventricular fibrillation or flutter fibrillation was emphasized in 1926 by DeBoer[2] and in 1929 by Davis and Sprague.[1] Six cases of a total of 14 with ventricular fibrillation reported up to 1929 were associated with heart-block. In two cases the abnormal rhythm was probably induced by digitalis, in one of these digitalis in combination with quinidin.

An examination of the reported cases of ventricular tachycardia also reveals evidence of bundle tissue disease, or the action of drugs that depress these tissues in a large number. Most of the reported cases were associated with the following conditions: (1) digitalis poisoning, (2) bundle tissue or bundle-branch disease, (3) coronary thrombosis. Rare cases are reported to have occurred in young adults with little or no demonstrable organic heart disease,[3, 4, 5, 6] in patients with rheumatic and syphilitic heart disease, and possibly as a result of excessive tobacco smoking.[7]

THE RELATION OF DIGITALIS TO VENTRICULAR TACHYCARDIA

In studying the progressive action of increasing doses of digitalis on the cat's heart, Robinson and Wilson[8] found the following sequence of events: inversion of the T-wave of the electrocardiogram; depression of the atrio-ventricular conduction with some slowing of the heart rate; increased rhythm of both auricles and ventricles with complete A-V dissociation, the ventricular rate soon exceeding the auricular. After complete A-V dissociation idioventricular complexes made their appearance, and independent ventricular rhythms with abnormal ventricular complexes occurred. This was followed by ventricular fibrillation and death. Luten[9] and others have shown that digitalis poisoning in man produces the same sequence of changes observed by Robinson and Wilson in cats. Commenting on an analysis of four cases in which ventricular tachycardia developed in the course of digitalis poisoning, he states, "It would appear, therefore, from a consideration of these four cases,

that toxic doses of digitalis produce on the human heart, when administered in increasing amounts, the following sequence of abnormal mechanism: auricular tachycardia; increase in the ventricular rate until it exceeds the auricular rate; and independent ventricular tachycardia with abnormal complexes." In his cases prolongation of the A-V conduction time was an early effect, and the increased ventricular rate always followed complete A-V dissociation. This association and sequence is significant, for it points to the importance of the state of bundle tissue in the course of development of the abnormal ventricular rhythm.

Vaughn[10] reported two cases of ventricular tachycardia associated with the administration of full doses of digitalis and considered it the probable factor responsible. He noted that although ventricular premature beats were frequent and the use of digitalis was common, runs of premature beats or tachycardia was rare. In 189 cases at the Peter Bent Brigham Hospital with the diagnosis of premature beats, he found but seven or 4 per cent with successive extrasystoles. Sixty-three per cent of the cases in this series had received digitalis, but in a large proportion digitalis administration was begun after the tracings were taken. In subsequent tracings no increase in the number of extrasystoles occurred. Vaughn concluded from this analysis that digitalis in therapeutic doses does not increase the tendency to successive ventricular extrasystoles. A similar conclusion was reached by Otto and Gold[11, 12] who felt that digitalis in full doses sometimes abolishes ventricle premature beats, and that premature beats coming spontaneously or induced by digitalis do not indicate susceptibility to digitalis bigeminy. Vaughn suggested that digitalis may be an exciting cause in the production of ventricular tachycardia but that some other factor such as hyperirritability resulting from impaired blood supply must be present. In one of the two cases reported by him bundle-branch block (P-R 0.20; QRS 0.12) was present before the administration of digitalis, and in this case it is probable that two factors were operative: (1) the underlying disease of the bundle tissue; (2) the depression of these tissues by digitalis. The conclusions by Vaughn, and Otto and Gold regarding the relation of normal doses of digitalis to premature beats lends further weight to this interpretation.

Tachycardia of ventricular origin associated with full or toxic doses of digitalis has been reported by many observers including Gilchrist[7] (two cases), Schwensen[13] (two cases), Felberbaum[14] (one case), Reid[15] (five cases), Levine and Curtiss[16] (one case), Gallavardin[17] (one case), Howard[18] (one case), Palmer and White[19] (two cases), Marvin[20] (five cases), and Strauss[21] (two cases). In one of Gilchrist's cases there was evidence of pre-existing disease of the bundle tissue with a P-R interval of 0.31 before the administration of digitalis. Smith, Schwensen and Gilchrist stressed the action of digitalis on the ventricular muscle as the precipitating factor. Smith thought that the action of digitalis

was variable and that the state of the heart muscle must be the deciding factor; Schwensen, that digitalis probably induces irritability in the ventricles; and Gilchrist, that the action of digitalis on a malnourished ventricular muscle might be the combination of factors responsible. In most of the reported cases in which there are tracings of the normal rhythm before and after the onset of the tachycardia, a marked prolongation of the P-R and often of the QRS interval is to be observed. Smith noted that of 60 reported cases, digitalis had been administered and was probably the responsible factor in 25. Strauss recorded digitalis therapy in 50 per cent of 65 cases reported up to 1930. Three out of a group of six cases with this arrhythmia among electrocardiographic records of the Beth Israel Hospital during the years 1928-1932 were definitely associated with excessive doses of digitalis. In one of these there was evidence of underlying bundle tissue disease.

THE RELATION OF BUNDLE TISSUE DISEASE TO VENTRICULAR TACHYCARDIA

As records of the normal electrocardiogram, either before or sufficiently after the administration of digitalis were not reported in all cases, it is impossible to obtain an exact idea as to the frequency of pre-existing bundle tissue disease in the digitalis cases. If conduction disease of the main bundle (P-R interval) had been present, this would be obscured in many, as the P-R interval was seen to be greatly increased in the postparoxysmal records. However, evidence of pre-existing bundle-branch disease was noted in 5 instances in the group receiving digitalis. The cases of Vaughn and of Gilchrist were mentioned above. In one of Marvin's cases there was pre-existing bundle-branch block with a P-R interval of 0.20 and a QRS interval of 0.12 seconds. Necropsy examination of the bundle branches in serial section showed definite disease in one bundle, thus confirming the electrocardiographic findings. Bundle-branch disease may also have been present in one of Palmer and White's cases, the QRS interval measuring 0.12 seconds.

One case reported by Butterfield and Hunt[22] occurred in a man aged forty-five years with rheumatic heart disease. The electrocardiogram showed a P-R interval of 0.18 and a QRS interval of 0.13 seconds. Postmortem examination revealed thickening of both mitral cusps, lymphatic infiltration, and an increased amount of connective tissue in the region of the bundle. The chief changes were found in the region of an antemortem clot adhering to the left side of the septum, and here an active pathological process was in progress at the time of death. The most important changes involved two-thirds of the thickness of the interventricular septum and consisted of a progressive fibrosis involving the muscle to such an extent that very few cardiac fibers could be recognized as such throughout a considerable amount of tissue. The increase in the QRS interval in this case was due either to involvement of the bundle branches or conceivably to block in the spread of excitation shortly after leaving the bundle branches in the

septum. Hart[23] reported one case in 1912 with unquestionable ventricular tachycardia. The interparoxysmal records show a P-R interval of 0.25, a QRS interval of 0.15 seconds. At the time of the paroxysm, the patient was on a small amount of digitalis, but when digitalis was out of the system the evidence of bundle-branch block was present. One case reported by Willius showed a P-R interval of 0.25, and a QRS of 0.10 seconds. In another case, in the same author's series, evidence of so-called "arborization block" is present. In Porter's[24] case associated with an attack of coronary thrombosis, the P-R interval was 0.22, the QRS interval 0.12 to 0.13 seconds. There was no indication of the use of digitalis. In one of Levine's cases[6] of ventricular tachycardia complicating coronary thrombosis definite bundle-branch block was present.

Two cases taken from the records of the Beth Israel Hospital showed ventricular tachycardia associated with bundle disease in the absence of digitalization. One case presented partial A-V block with P-R intervals as long as 0.42 seconds. In this case the paroxysmal ventricular complexes differed but slightly from the normal, suggesting the origin to be in the main bundle tissue. The second case occurring in a patient with coronary thrombosis in the past showed a P-R interval of 0.21, a QRS interval of 0.12 seconds.

THE RELATION OF VENTRICULAR TACHYCARDIA TO CORONARY THROMBOSIS

A number of cases of ventricular tachycardia are now on record as complicating an attack of acute coronary thrombosis. In most of these cases the conduction time in the tracings preceding and following the paroxysm is normal. It is known that the septal muscle receives its excitation slightly in advance of that of the outer walls of the ventricles and less than one-tenth of a second is required before all parts of the muscle are in a state of activity. For practical purposes this distribution is nearly simultaneous. The slight difference, however, in the time from the moment the excitation arrives at one place and is finally distributed at all points is probably represented by the QRS interval and when this is normal it is probable that there is little gross interference with the His-Tawara-Purkinje distribution. Yet for the occurrence of ventricular tachycardia in a condition in which the function of varying areas of heart muscle is disturbed, and in a condition in which the septum is often the site of involvement (the anterior descending branch of the left coronary being frequently involved), an explanation must consider the possibility of temporary disturbances in the conduction tissues. Although an infarct may not interfere with the endothelial transmission of impulses in the Purkinje network, there may be moments when the development of local edema or mural thrombosis causes such interference. The presence of large infarcts or mural thrombi in many of the cases developing ventricular tachycardia following coronary thrombosis is suggested by the outcome in Levine's series of 8 cases. Six died, in spite of the fact that the tachycardia was

regularly controlled by quinidine. It therefore seems reasonable to consider a factor of this kind in addition to the factor of local hyperirritability in the muscle.

THERAPEUTIC CONSIDERATION

The action of quinidine in the abolition of attacks of ventricular tachycardia has been recorded in many cases. Lewis and his coworkers[25] studied in the action of quinidine in dogs. An early and marked effect was produced in the heart muscle, and this consisted in a slowing of the rate of conduction and an increase in the refractory period. The action on the conduction tissues was also striking, a lengthening of the P-R and QRS intervals being regularly noted with sufficient dosage. There is evidence that in man a powerful action may be exerted on the muscle before an appreciable effect on the conducting tissue is noted. Thus, after the abolition of auricular fibrillation in clinical cases, the electrocardiogram often shows no appreciable increase in the P-R and QRS intervals. It is therefore probable that quinidine exerts its action on the ventricular muscle to the extent of interrupting a tachycardia without causing significant depression of the bundle tissues in most cases. In rare instances and when bundle tissue disease is present, quinidine may precipitate fibrillation of the ventricles by exerting a predominant action on the conducting tissue. This probably occurred in the case of Kerr and Bender.[26]

On the basis of the above, it would seem that digitalis therapy should be undertaken with caution in the presence of bundle tissue disease. Drugs which tend to increase conduction might be considered in the attempt to stop a paroxysm of ventricular tachycardia. Atropine, by diminishing vagal action may be of value. Levine[27] has had one success with this drug. On theoretical grounds, adrenalin, ephedrin, barium chloride and thyroid extract might be of value, in sufficient dosage. When combined with quinidine the untoward effect of these drugs on the ventricular muscle might be controlled.

SUMMARY AND CONCLUSIONS

1. An interpretation of the mechanism of ventricular tachycardia is presented: Its occurrence in healthy individuals is normally prevented by the control effected through the His-Tawara-Purkinje system of conduction, which distributes supraventricular impulses very rapidly to all parts of both ventricles and induces a uniform state of refractoriness sufficient to prevent the setting up of continuous runs of ectopic beats. Disease or drugs that depress the conducting tissues tend to interfere with this normal control and permit the development of circus movements in the ventricles.

2. An analysis of the literature reveals the presence of disease or the use of drugs which depress the conducting tissues in a majority of the reported cases.

3. Ventricular tachycardia resulting from digitalis is probably induced by its depressing action on the conducting tissues, and this factor must be considered together with hyperirritability of the muscle.

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CARDIAC RUPTURE WITH PERFORATION OF INTERVENTRICULAR SEPTUM. REPORT OF TWO CASES*

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RUPTURE of the heart is moderately frequent following thrombosis, but perforation is usually through the wall into the pericardium, rupture of the septum being exceedingly rare. The event of interventricular rupture is accompanied by sufficiently characteristic symptoms and signs to make the condition recognizable ante mortem. There are, however, certain similar conditions that have to be differentiated, namely, external rupture of the heart allowing blood to pass through into the pericardium, and ruptured aneurysm of the right anterior aortic sinus causing a communication between the aorta and the base of the right ventricle. The latter condition may be of congenital origin, the clinical evidences of its presence existing throughout life, but in far the greater number of cases (Abbott) it results from rupture of an aneurysm of the aorta into the pulmonic circulation.

The cases of spontaneous cardiac rupture collected by Krumbhaar and Crowell showed the following distribution, the great majority being through the wall of one of the cardiac chambers into the pericardial cavity.

TABLE I

SITE OF SPONTANEOUS RUPTURE (KRUMBHAAR AND CROWELL)

	TOTAL	PER CENT
Right auricle	35	5.7
Left auricle	12	2.0
Right ventricle	63	10.2
Left ventricle	493	79.7
Miscellaneous*	15	2.4

*The miscellaneous group consists mostly of ruptured papillary muscles but includes the two cases of rupture reported by Latham.

Three typical cases of interventricular rupture have previously been reported. Abstracts of these cases are given.

CASE 1. (Latham.) White male, aged sixty-one years, of gouty diathesis and sedentary habits, complained of unusual pain occupying entire front of chest and passing along both clavicles to the top of the shoulders, stopping there without descending the arms. Pain remained constant for two days but became more severe during the last night. The pulse was normal in rate but hard and incompressible as it had been for a long time. A mustard plaster was applied and some cordial aperient given. No relief was obtained next day, the patient having been sleepless and unable to lie down. The heart beat over a larger space with more force than natural but with no unnatural sounds. The pain was like that of angina pectoris but continuous. One-half dram of vinum colchici and one dram of paregoric were

*From Saint Elizabeth's Hospital.
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administered and repeated in six hours. The next day the patient was quite free from pain, and the medication was discontinued because of nausea. The patient remained comfortable for two days and two nights whereupon the pain returned, the patient became deadly pale, the entire body was cold as marble and streaming with perspiration. The pulse was of good strength, the heart contracting regularly and forcibly, and now for the first time a loud systolic murmur was audible over the precordial region. Large doses of opium with ether and ammonia were administered, but after two hours the pain remained unabated. The pulse began to sink and the patient looked and felt like a corpse. Ten hours after the onset of pain the patient became pulseless but lived seven hours longer with mind clear to the end.

At postmortem examination, "the heart was a good deal larger than natural and incased in fat." The muscular substance was very flaccid and thin, becoming thinner as the apex was reached where it was reduced to a mere line, but healthy in color. "The capacity of the right ventricle was notably larger than natural. Its internal lining was stained a deep red. Its orifices were free and its valves healthy." There was left ventricular hypertrophy, the muscle of healthy color; "but in the septum it was pale and soft, manifestly in consequence of fatty degeneration. In the septum, at its posterior juncture with the parietes, there was an oblique rent passing through it from ventricle to ventricle. On the side of the left ventricle it was an inch and a half in length; on the side of the right, it just opened at a point. The orifices of the left, as of the right ventricle, were quite free, and its valves healthy. The coronary arteries contained some atheromatous deposits, but were quite pervious. The aorta was healthy as far as its arch. It began to be studded with atheromatous and earthy matter in its descending portion."

Case 2. (Latham.) The pathological specimen of this case was discovered by Latham in the museum of St. Bartholomew's. Latham ascertained the circumstances of death and clinical history from various sources. Male, sixty years of age, "inclining to corpulence, had for several years been liable to occasional attacks of severe inflammation of lungs, requiring copious bloodletting for their cure." In February, 1829, having had such an attack during the previous winter he began to experience a new complaint, namely, "pain beneath the sternum, suddenly coming and going, attended by pain or numbness down one and sometimes down both arms, and by something more than pain, an indescribable anguish, generally within the chest." The attack would seize him as he walked along the streets making it necessary to stop and rest. Near the end of April "he was suddenly seized with pain beneath the sternum and down both arms. It was severe and agonizing beyond what it had ever been before." He looked like a dying man, pale and purple about the lips and had a very frequent and hardly perceptible pulse. By the help of stimulants he was kept alive three days.

The description of the heart as described in the catalogue of the museum follows:

"The cavity of the right auricle is found larger than natural and its membranous lining thick and opaque. The tricuspid valve is thickened, and its lining opaque. The aortic valves are a little thickened, and there is a soft matter beneath the lining of the aorta just above the valves. The coronary arteries are thickened, and there is bony matter deposited between their coats. The rupture of the septum dividing the ventricles has taken place near its union with the posterior wall of the heart, by which a free communication is made between the two ventricles. On the side of the left ventricle the opening is about two inches in length and of a semilunar form. On the side of the right the opening is much smaller and rounded."

Case 3. (Hyman.) Male, white, aged fifty-three years, previously enjoying good health and of rugged constitution, awakened suddenly one morning with severe cramplike pain in the lower abdomen. He felt nauseated and vomited several times. The pain then became localized in the epigastric region and reached such an intensity that the patient rapidly became prostrated. Diagnosis of ruptured

duodenal ulcer was made and operation advised. Relatives objected and two hours later another physician noted the patient's desperate condition and very feeble heart action. Four hours after the onset the patient was seen by Hyman. The patient was now in extreme shock with cold, clammy perspiration, deeply cyanosed and breathing with noisy respirations. There was no palpable pulse at wrist, and the superficial area of cardiac dullness appeared widened to percussion. No heart sounds could be heard at the apex but the basal sounds were rapid (130 per minute) and of very poor quality. There was a very shrill systolic murmur heard equally well over the entire precordia. A diagnosis of spontaneous rupture of the heart was made and hospitalization advised. The patient was admitted to the hospital at 11:30 A.M., electrocardiographic studies were made and intravenous glucose and metaphyllin administered. The heart sounds now became more audible at the apex as well as at the base, but the superficial character of the murmur remained unchanged. The patient died half an hour later, death occurring five and one-half hours after the initial attack of pain.

Electrocardiographic studies showed characteristic T-wave alterations in Leads I and II. There was no axial deviation of heart and no delay in the QRS complex. The rate was 100 beats per minute. Sino-auricular rhythm was normal.

At postmortem examination the pericardium was found free from blood. The heart was slightly enlarged (310 grams), and there was a recent perforation large enough to admit one finger in the interventricular septum near the apex. The coronary arteries contained irregular patches of sclerosis; the right was more involved than the left. A rather large thrombus which had apparently broken off from an adjacent area was found in the terminal subdivision of the anterior descending branch of the right coronary artery. The portion of the heart supplied by this vessel was pale, thinned out, and the change appeared to be not very recent. The apical part of the heart, especially in the right ventricle, contained a large hemorrhagic area which was torn through as it approached the septum. Several mural thrombi were found in the left ventricle. The valve mechanism presented nothing of note, and the aorta was not enlarged but showed many atheromatous plaques.

In addition to the cases of septal perforation following myocardial infarction, communication between the two ventricular cavities or between the aorta and the base of the right ventricle may develop in a somewhat different manner.

Abbott's study of abnormal communications between the aorta and the base of the right ventricle or between the aorta and the pulmonary artery is interesting because of the similarity of the symptomatology to that of our cases and the difference in pathology. She points out that a communication may arise in two ways: (1) as the result of a ruptured aneurysm where the wall of the ascending aorta is extensively diseased from luetic or other causes; (2) when the right anterior aortic sinus is the seat of a finger or thimblelike process which projects into the conus of the right ventricle. This constitutes an aneurysm of its wall and that of the aortic sinus due not to disease but apparently to congenital thinning of the septum between the two great trunks. This latter explanation may be applied to the cases of Beck, Hale-White, Krzywicki and Kraus in which the walls of the aneurysm were thin and membranous without sign of inflammatory process of any kind and rupture had taken place at the apex of the sac, evidently as a result

of strain. In the case of Krzywicki the ventricular septum was entire, but in the other above cited and in several similar ones in the literature (Hart) (Thurman) (Abbott) in which the conditions were more or less obscured by the existence of malignant endocarditis, the aneurysm of the right aortic sinus was associated with a defect of the anterior interventricular septum immediately below the cusp. Both of these conditions have been considered by the authors as due to defective development of the aortic (bulbar) septum. Richards and Charteris have reported similar defects. Abbott and others believe that aneurysm of the right aortic sinus and the associated interventricular communication are of congenital origin because of their location immediately above and below the right aortic cusp, of the shape and appearance of the septal opening as seen from the left ventricle, supported by the facts of developmental and comparative anatomy.

We have had the opportunity to observe two cases of perforation of the interventricular septum following coronary occlusion.

CASE REPORTS

Case 1. White male, aged sixty years, a custodial case since 1901 in Saint Elizabeth's Hospital, was well nourished and well developed and enjoyed fair health until 1927. Since then he had grown progressively weaker, finally necessitating confinement to a wheel chair in June, 1930. There was no noticeable change in his condition until April 9, 1931, when he was hospitalized because of what appeared to be an acute respiratory disorder. The lips, fingernails, and feet were slightly cyanotic. The face was flushed, temperature 102 degrees, pulse 100, respiration 26, dyspneic. The cyanosis quickly disappeared upon rest in bed. The chest showed no impairment of percussion note, and auscultation revealed moist râles at both bases posteriorly. The cardiac rhythm was regular and there were no murmurs. Blood pressure, systolic 102 mm., diastolic 68 mm. The peripheral arteries were beaded and tortuous. In the absence of other positive findings the condition was diagnosed as acute upper respiratory infection. The temperature returned to 100 degrees on the second day and remained at that level until the terminal process.

Blood count revealed: hemoglobin 81 per cent, W. B. C. 16,200, morphonuclears 76 per cent. The patient was placed on appropriate treatment for a respiratory infection and seemed to improve somewhat from day to day until 6 A.M., April 16, when he suddenly had a sinking spell while his bed was being made up. Respiration became rapid and shallow, the pulse rapid and weak; the skin was cold, clammy and pale and covered with great beads of perspiration. He appeared to be in shock. A loud, shrill systolic murmur now appeared at the apex and was audible over the entire precordium. One hour later the pulse could not be counted at the wrist and remained absent during the rest of his life. Later that day the patient developed Cheyne-Stokes respiration and a significant change in the character of the cardiac sounds. The systolic murmur had increased in intensity, and the aortic second tone which had been present before was no longer audible. The liver could now be palpated two finger breadths below the costal margin. As the day wore on the murmur became louder and audible over the entire chest. A marked systolic thrill developed, and the murmur became so harsh and so superficial that it could easily be heard by placing the ear near the chest. It was particularly noted that though the apical impulse and action of the heart were very strong, the pulse was absent in both wrists. This seemed to indicate that something had occurred which interfered with the forward propulsion of the blood. Something in the nature of

acute aortic stenosis was considered, such as the formation of a mural thrombus in the left ventricle obstructing the aortic outlets of that chamber. Other diagnoses considered included ruptured aortic leaflet and also the formation of a dissecting aneurysm. Ruptured aortic leaflet was not considered seriously on account of the fact that this would not obstruct the forward propulsion of blood. The next day the patient developed evidence of a mild hypostatic pneumonia, grew gradually weaker, and died that night, April 17, at 8:30 P.M. A final diagnosis of coronary thrombosis and hypostatic pneumonia was given.

Electrocardiographic studies showed tachycardia, left ventricular preponderance and a convex inverted T-1. The manner in which the T-wave took off below the base line in Leads II and III suggested coronary occlusion (Fig. 1).

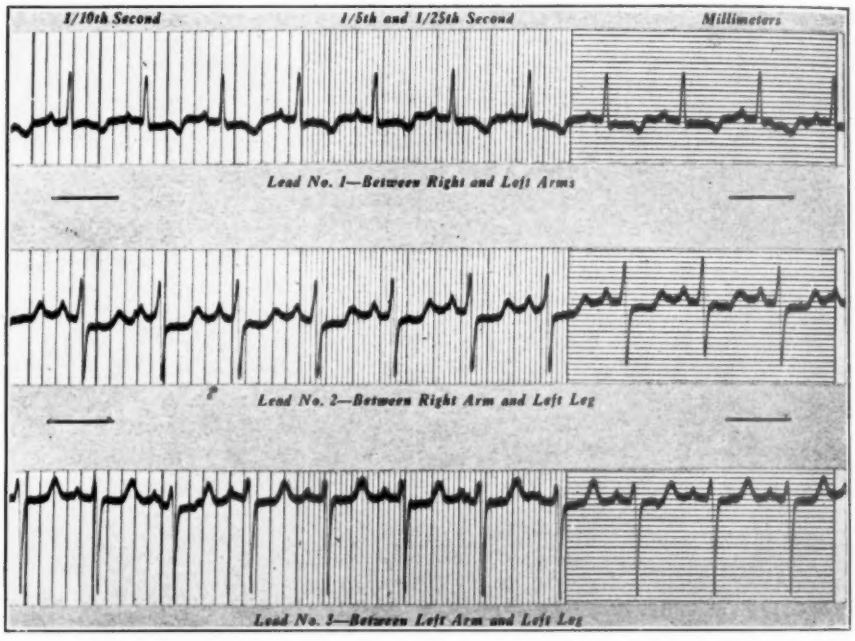

Fig. 1.—(Case 1.) Electrocardiogram taken April 16, showing left ventricular preponderance and a partial "coronary T-wave."

Necropsy disclosed generalized arteriosclerosis with nephrosclerosis and multiple infarcts of the brain; passive congestion of all the organs and intense congestion and edema of the lungs without pneumonic consolidation or pleural effusion. The heart weighed 545 grams. The pericardial cavity contained very little fluid. The right ventricle appeared particularly dilated and was filled with partially decolorized clots. The wall was rather pale but normal in consistency and texture. The right atrium was distended with partly decolorized clots and the auricle contained an adherent thrombus. Dark, soft, currant-jelly clots were found in the veins leading into the right atrium. The left ventricle was thickened posteriorly but was thin and rather fibrosed anteriorly. The myocardium had a rather dull red beefy color, was quite fine in texture and showed little or no fibrosis externally. The left atrium was not particularly dilated and its appendage was collapsed. There were mixed clots in both atrium and ventricle, and soft red clots in the pulmonary veins. The wall of the left ventricle measured 25 mm. in thickness, the right 5 mm., the circumferences of the valvular orifices were as follows: aortic 8.5 mm., mitral

10.5 mm., pulmonic 9 mm., tricuspid 14 mm. The valves on the right side of the heart were quite normal in character, although the tricuspid orifice seemed dilated; the mitral valve was slightly thickened and sclerotic but fairly flexible. The leaflets of the aortic valve were definitely thickened, showed bits of calcareous material at their bases and projected rather rigidly into the lumen, but not sufficiently to cause obstruction to the forward flow of blood. They were competent to the water test. The coronary arteries showed marked atheromatous degeneration with calcification. Wherever one of these was cut through with the scissors or knife, there was a crunching or grating sound. The changes were most marked in the anterior descending branch of the left coronary artery which was thicker in parts than a lead pencil and which showed almost complete obliteration of the lumen. On account of

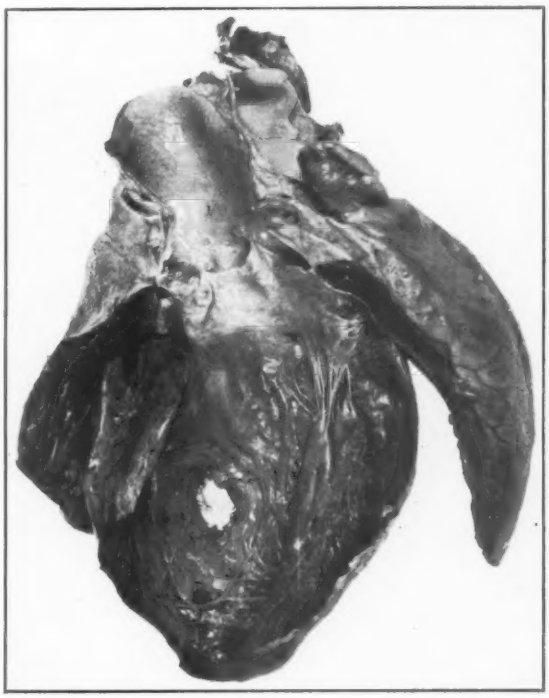

Fig. 2.—(Case 1.)—Photograph of the heart showing septal perforation in the middle of an aneurysmal bulging.

a desire to preserve the specimen it was not dissected free. The other coronary branches were sclerotic to a lesser degree.

Upon opening the left ventricle there was found an area of dilatation in the lower part near the septum, with separation of the pectinate muscles and columnae, forming an aneurysm 7 to 8 cm. in diameter, the walls of which were smooth, flat and covered by thick whitish endocardium. In the center of this was a much deeper depression, dark in color, with somewhat ragged torn edges and a little reaction surrounding it. The tip of the index finger passed easily through this perforation into the cavity of the right ventricle within 2 cm. of the tip of the latter. Rather intimately applied to this, on the right side there was a thick membrane made up of fibrin and fresh thrombus, attached to the lateral wall of the right ventricle and partially covering the opening. Some of the columnae carneae were not yet broken down in this vicinity and gave added foundation for the thrombus. When the heart

was held up to the light and the columnae carneae retracted, it was easy to see from one ventricle into the other. There was a moderate bulge of the septal wall into the right ventricle, most marked in the region of the perforation (Fig. 2).

CASE 2. White female, aged seventy years, a resident of various mental institutions since 1904, had good physical health until 1914 when she was operated on for hernia. In 1915 she fractured her right hip and was very lame for the rest of her life. During 1926 she suffered somewhat with bronchitis and laryngitis and in 1929 was hospitalized for a time because of dysuria and hematuria and again in 1930 for herpes zoster which was followed by severe anemia. Suddenly during the night of June 1, 1931, the patient developed acute abdominal pain necessitating the use of morphine. On examination the following morning the patient was fully conscious

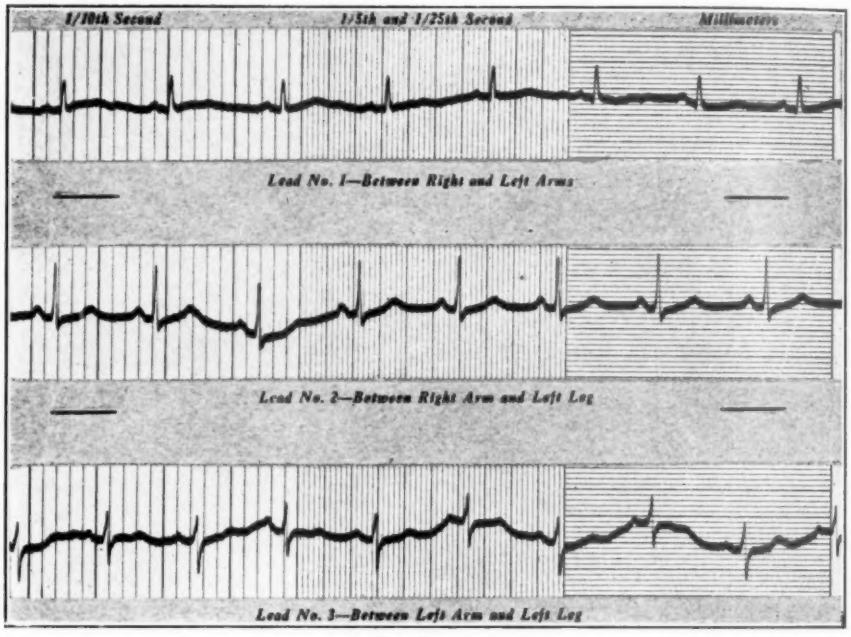

Fig. 3.—(Case 2.) Electrocardiogram taken June 2 showing partial "coronary T-wave" in Leads II and III.

and complained of severe intermittent pain of a stabbing character which seemed to be localized across the upper abdomen and under the left costal margin. She also complained of pain in the left arm and upper interscapular area. The patient yawned occasionally. Her skin was pale and she appeared to be in shock. Morphine was administered but the discomfort and pain were not entirely relieved. There was no cyanosis of any part of the body, but slight edema of the ankles was present. Examination of the chest revealed no impairment of percussion note over the lungs. There were numerous coarse râles over both bases posteriorly, more marked on the left side. The area of cardiac dullness was not appreciably enlarged and auscultation revealed regular cardiac rhythm and no murmurs. The peripheral arteries were slightly tortuous. Blood pressure was systolic 150 mm., diastolic 60 mm. The liver was not palpable. Diagnosis of an acute coronary occlusion was made. Electrocardiogram substantiated this by the low take off of T-2 and T-3 (Fig. 3).

The patient continued to suffer the above described pain for two days and vomited occasionally. On the third day her temperature became elevated and continued so

until her death. On the fourth day of her illness examination revealed a well-marked systolic thrill, and a loud systolic murmur over the entire chest and a systolic blood pressure of 80 mm. The thrill and murmur became more pronounced on the following day. The patient grew gradually weaker and died June 7, 1931, at 2:12 P.M.

A final clinical diagnosis of coronary thrombosis and generalized arteriosclerosis was made.

Necropsy disclosed generalized arteriosclerosis with passive congestion particularly marked in the lungs and associated here with thrombosis and multiple hemorrhagic infarcts. The kidneys showed arteriosclerosis with multiple infarcts and a hypernephroma invading the pelvis on the left side. The gall bladder was contracted

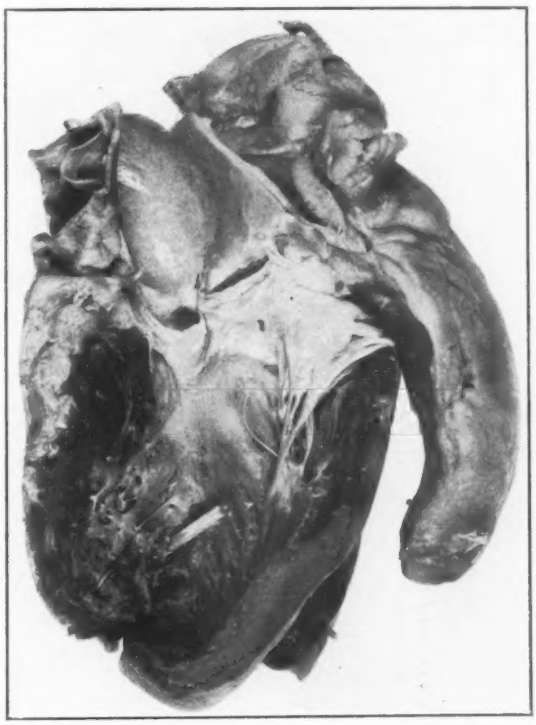

Fig. 4.—(Case 2.) Photograph of the heart showing a match inserted into the interventricular perforation.

around two stones; there was a colloid cyst of the thyroid; the brain was atrophic but not particularly arteriosclerotic.

The pericardial cavity contained a small amount of dark amber fluid. The heart weighed 435 grams, but appeared much larger owing to marked dilatation. The epicardium was smooth and thin, rather liberally supplied with fat. The right atrium was markedly distended with mixed clots, but no thrombi were found in the appendage; the tricuspid orifice appeared normal and the leaflets were thin and flexible. The right ventricle contained fairly firm, partially decolorized clots that became more adherent in the lower portion, especially to the septum. The lateral wall of the right ventricle was of good color and consistency, but the septum appeared pale and soft, with hemorrhagic areas here and there. A small perforation was found within about 2 cm. of the apex. This admitted a match (Fig. 4), although

the area of ragged laceration was somewhat larger and definitely greater on the side of the left ventricle. The area of anemic and hemorrhagic necrosis measured about 4 cm. in diameter, and there was a thinning of the septum around the point of perforation, but very little aneurysmal bulging was observed. The apex of the left ventricle was dilated in aneurysmal fashion, with a thin, somewhat fibrous wall, and thickened endocardium, but organized thrombi were not found on this side of the heart. The perforation was partly sealed off by the adherent thrombi and clots on the right side of the heart. The valves showed no abnormalities. The coronary arteries were rather tortuous and showed atheromatous patches, especially marked in the anterior descending branch, but the location of the thrombus was not further investigated (Fig. 4).

DISCUSSION

Interventricular rupture like other ruptures of the heart occurs in middle or old age and is preceded by coronary disease with occlusion of a branch supplying the interventricular septum. Clinically the rupture is usually preceded by an acute illness in which all the symptoms may point toward some grave abdominal condition, as in Hyman's case where a diagnosis of ruptured gastric or duodenal ulcer was suggested and in our second case where the first complaint was of severe abdominal pain. Or the symptoms may at first simulate a respiratory disorder with dyspnea, cyanosis, fever and leucocytosis, as in our first case. Finally, the symptoms may attract attention to the heart from the beginning by severe pain in this region which may or may not radiate down the arms, as in Latham's two cases. In this initial period no diagnosis of septal perforation can be made whether or not the attention is attracted to the cardiac mechanism. Examination of the heart at this time may be entirely negative or may indicate anginal failure. Electrocardiographic tracings may indicate coronary occlusion or myocardial fibrosis. Murmurs are usually absent, and the history usually shows fair health prior to the acute onset. The persistence of the symptoms, however, especially the pain, reveals the seriousness of the patient's condition, and the striking change in the patient and clinical findings following the rupture should make this condition distinguishable from other complications.

Perforation of the interventricular septum is ushered in by renewed syncope, by an increasingly harsh systolic murmur and thrill with gradual extinction of the aortic second sound, progressive fall in blood pressure with obliteration of the pulse, ashen pallor rather than cyanosis, and pulmonary inundation. The disproportion between the force of the cardiac impulse and the quality of the pulse, together with the increasing thrill and murmur and the absent second sound would appear to be particularly suggestive of septal perforation.

The elapsed time from the onset of symptoms until rupture of the septum, according to a survey of the five cases at hand, varies from two days to six weeks.

Rupture of the heart into the pericardium usually causes instantaneous death, or the patient may go into profound shock and die in a few hours.

The length of life is somewhat longer, however, following interventricular rupture as shown in Table II.

TABLE II

LENGTH OF LIFE AFTER INTERVENTRICULAR RUPTURE

Latham's case	No. 1	17 hours
Latham's case	No. 2	3 days
Hyman's case		5½ hours
Our case	No. 1	38½ hours
Our case	No. 2	1½ days

Rupture of an aneurysm of the right anterior aortic sinus of Valsalva occurs suddenly in a person in apparently perfect health, and the patient may survive the rupture for years, the accompanying precordial thrill and murmur persisting unchanged. Furthermore, the rupture of such aneurysms occurs in relatively young individuals. The symptoms in such individuals are usually less serious because of the better condition of the musculature of the heart. The sudden diverting of part of the blood stream from the systemic circulation through the septum into the lesser circulation is followed by considerable strain upon the musculature of the right ventricle. A healthy musculature will be able to compensate for this added load, and the heart will continue to function in a satisfactory manner; but if the muscle is already diseased, congestive failure develops in the lesser circulation.

The location of the perforation itself probably has little effect upon the electrocardiographic tracing, but it is interesting that no more serious or specific changes are present. The location of the septal infarction would appear to be sufficiently distal to permit the main conduction bundles to escape. The higher in the septum the infarct extends, the more profound are the observed electrocardiographic alterations.

The rarity of septal perforation in comparison with external perforation would seem to be due to the relative freedom of the septum from marked changes in the pressure applied to the two sides. While the pressure within the left ventricle probably rises to some 200 mm. Hg in the normal heart, this is compensated on the other side of the ventricle by a pressure probably half as great. In cases of coronary occlusion there is undoubtedly a distinct fall in the intracardiac pressure in both chambers, but the ratio of left to right would probably show less pronounced alterations. Thus the integrity of the septum would tend to be preserved. Indeed, infarction of the septum is by no means rare, but its perforation would appear to be almost a curiosity.

The clinical picture of pallor, pulselessness, Cheyne-Stokes respiration, absence of the aortic second tone, in conjunction with a loud systolic murmur which is out of all proportion to the character and intensity of the other heart sounds and a forceful action of the heart indicates that there is something preventing the forward propulsion of the blood. In

view of the pathologic condition it is easy to see that the development of shock with deficient circulation, a loud systolic murmur and thrill, and no pulse, would result from blood in the left ventricle being forced through the septum into the right ventricle through a roughened orifice. This is brought about by the fact that the pressure is physiologically highest in the left ventricle. Pallor and shock rather than cyanosis result because the blood is propelled into the lungs from both chambers of the heart and accumulates there rather than in the body, but is essentially well aerated.

The symptomatology of ruptured aneurysm of the right anterior aortic sinus of Valsalva, leading to communication between the aorta and the base of the right ventricle as given by Abbott, shows many points of similarity, including dyspnea without cyanosis, precordial vibration and thrill of intense purring character, loud sawing murmur, etc. This condition as well as pure ventricular rupture may be differentiated from patent ductus by the extremely superficial character relative to the chest wall and by the location and intensity of the sounds. A history of marked pulsation in the temples and vessels of the neck, dyspnea on exertion without cyanosis or edema, precordial vibration and a strong diastolic thrill over the precordium, a systolic thrill in vessels of the neck, a loud rough diastolic murmur at the base with systolic and diastolic murmur at the apex as in Abbott's case may indicate that the patient is suffering from a ruptured aneurysm of the right anterior sinus of Valsalva, especially if the patient is relatively young. Such a condition may be compatible with life for years. This is not so with ruptured interventricular septum which is always a terminal process and occurs in old age.

The symptomatology of other cases of cardiac rupture is quite different. Where the rupture is into the pericardium, the patient is seen to fall over dead or is found dead. If the patient survives for a short time, the symptoms are those of acute collapse with pallor, air hunger, marked cyanosis, stertorous breathing, cold sweat and sometimes convulsions or unconsciousness. Prior to the rupture the patient may have had cough, dyspnea, vertigo, syncope, hematemesis, anginal pain, diarrhea, vomiting, etc., namely, the signs of coronary disease.

SUMMARY AND CONCLUSIONS

1. Two cases of perforation of the interventricular septum following coronary thrombosis are reported.

2. This rare condition should be suspected when there is superimposed upon the picture of coronary occlusion a striking symptom complex consisting of (a) progressive harsh systolic murmur and thrill over the precordium, (b) gradual extinction of the aortic second sound, and (c) marked disproportion between the force of cardiac action and the strength of the pulse.

3. The mechanism seems plainly indicated. The septal perforation permits passage of blood from the stronger left ventricle to the weaker right through a gap with rough edges reducing the stream that passes the normal aortic opening into the general circulation. Death may take place from pulmonary congestion and edema before the opening is large enough to divert sufficient blood from the systemic circulation to cause disappearance of the pulse.

4. The condition may be distinguished from congenital malformation by the age and previous condition of the patient as well as the shorter survival period.

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THE BLOOD PRESSURE AND ELECTROCARDIOGRAM IN EXPERIMENTAL PERICARDIAL EFFUSION*

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A VERY detailed account of the blood pressure changes in dogs during experimental pericardial effusion was given by Cohnheim in his "Lectures on General Pathology."[1] Oil was injected into the pericardial sacs of medium sized dogs and the pressure produced measured by an oil manometer. Kymographic tracings of the blood pressure in the femoral and pulmonary arteries were made with mercury manometers, while the venous pressure was recorded by a sodium carbonate manometer attached to the external jugular vein. The concentration of sodium carbonate solution used was not stated. Cohnheim found that an intrapericardial pressure of 30 to 40 mm. of oil caused a slight rise in venous pressure. An oil pressure of 60 to 70 mm. produced a fall of 20 to 30 mm. Hg in the arterial pressures and a rise of venous pressure to about 60 mm. of sodium carbonate solution. When the intrapericardial pressure reached 100 to 120 mm. of oil, the pulmonary and femoral pressures fell to about one-half of their original values, and the respiratory and systolic fluctuations of the mercury manometers were decidedly damped. The venous pressure rose to about 100 mm. of sodium carbonate solution. Further increase of pressure in the pericardium reduced the arterial pressure almost to zero, the pulse pressure disappearing first in the pulmonary and a little later in the femoral artery. The jugular pressure increased greatly. Usually an oil pressure of about 240 mm. was sufficient to reduce the pressure in the pulmonary artery to a minimum, while the femoral tracings still showed slight pulsations. The manometer in the external jugular vein then recorded more than 220 mm. of sodium carbonate solution. This condition of minimal arterial pressure could be maintained for two to three minutes without causing serious cardiac damage. On reducing the intrapericardial pressure the pulse appeared first in the pulmonary artery. The pulse waves were at first large and infrequent but soon returned to normal size. The arterial pressures rose rapidly to their normal values and usually slightly higher, while the venous pressure fell to zero. Cohnheim noted that if an increased intrapericardial pressure were maintained for a few minutes there was a gradual stretching of the pericardium which, of course, led to a diminution of the pressure in the pericardial sac and to a slight rise in arterial and fall in venous pressure.

Recently Scott, Feil and Katz[2] reported a case of hemopericardium and another of purulent pericarditis in which changes occurred in the electrocardiogram similar to those observed in recent myocardial in-

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faretion, and in animal experiments they produced similar electrocardiographic changes by artificial pericardial effusions.[3]

At the suggestion of Professor Roger S. Morris we sought more complete records of the relationship between intrapericardial and arterial pressures and the electrocardiogram to determine, if possible, how great a pressure in the pericardial sac is necessary to cause obvious changes in the T-wave of the electrocardiogram.

Medium sized dogs were given hypodermic injections of ⅛ to ⅔ grain morphine sulphate and anesthetized with ether. A tracheal cannula was inserted for control of the anesthetic. The pressure in the right carotid artery was recorded by a mercury manometer. The chest was opened and artificial respiration established. The intrapericardial pressure was varied by introduction of saline through a suitable cannula and measured by a mercury manometer. Injection of drugs was made from burettes attached to cannulas in the femoral veins. Electrocardiographic tracings were recorded in Lead II, using the Victor apparatus. The times of taking these records were marked on the kymograph by an electromagnetic signal. Throughout these experiments the chest was kept open. The thoracic organs were protected from drying by a layer of paraffin.

Our kymographic records completely confirmed the data of Cohnheim. A gradual increase in the intrapericardial pressure caused a fall in carotid pressure. The pulse pressure diminished and finally the carotid pressure tracing became a straight line parallel to and almost touching the base line. If the increases in the intrapericardial pressure were made by stages, there were established definite levels of arterial pressure for each stage. These were maintained usually for a minute or more, but then a slight rise in blood pressure was caused by stretching of the pericardium. When the intrapericardial pressure was slowly but continuously increased, there appeared to be a definite mathematical relationship between the pressure in the pericardial sac and that in the carotid artery, which was expressed by the general formula

$$P_x = \frac{P_o}{m.e^{(ax + bx^2 + cx^3 + dx^4)}}$$

in which

P_o is the initial blood pressure (mean of systolic and diastolic pressures) in mm. Hg,

P_x is the mean blood pressure under an intrapericardial pressure of

x mm. Hg, and

a, b, c, d are constants for the particular experiment.

The observed and calculated blood pressures in a typical experiment are given in Table I. For this series $P_o = 92$; $m = 0.9828$; $a = +0.02321$; $b = -0.00737$; $c = +0.00135$; $d = -0.000015$.

With a decrease in intrapericardial pressure there was not an immediate response in blood pressure if this had been reduced to a mini-

TABLE I

INTRAPERICARDIAL PRESSURE MM. HG	MEAN CAROTID ARTERIAL PRESSURE MM. HG	
	OBS.	CALC.
2	91	91.8
4	90	88.2
6	83	83.0
8	70	71.9
10	57	56.7
12	40	40.9
14	27	27.1
16	17	16.6
18	10	10.1
20	6	5.3

mum. After a short pause the heart began to beat forcefully and usually in such a way as to produce violent oscillations in the carotid pressure tracing and sometimes also in the tracing of the intrapericardial pressure. The pulse became more rapid, arterial and pulse pressures increased, and soon the heart was restored to normal activity. Fig. 1 shows the whole cycle of pressure changes, while Fig. 2 is an example of the more violent oscillations in the blood pressure tracing on reduction of pressure in the pericardial sac.

These changes in the intrapericardial pressure could be repeated many times on the same animal without any appreciable change in the myocardium but with a gradual stretching of the pericardium. This stretching was indicated by the increasing volume of fluid which must flow into the pericardial sac to produce a given intrapericardial pressure.

The actual volumes of saline introduced into the pericardial sac were not usually recorded, but data from a typical experiment are shown in Table II. It will be seen, as noted by Katz and Gauchat,[4] that there is not a simple arithmetic ratio between the volume of the fluid introduced and the pressure caused by this fluid. The actual relationship between volume and pressure is expressed by the exponential formula

$$P = e^{aV}.$$

For the calculated data of Table II the value of a was 0.0165.

TABLE II

C.C. SALINE INJECTED	MEAN CAROTID PRESSURE MM. HG	INTRAPERICARDIAL PRESSURE MM. HG	
		OBS.	CALC.
0	96	0.0	0.0
25	95	1.5	1.5
50	90	2.5	2.3
75	78	4.0	3.5
100	64	5.5	5.2
125	28	8.0	7.9
150	10	12.0	11.9
175	2	18.0	18.0

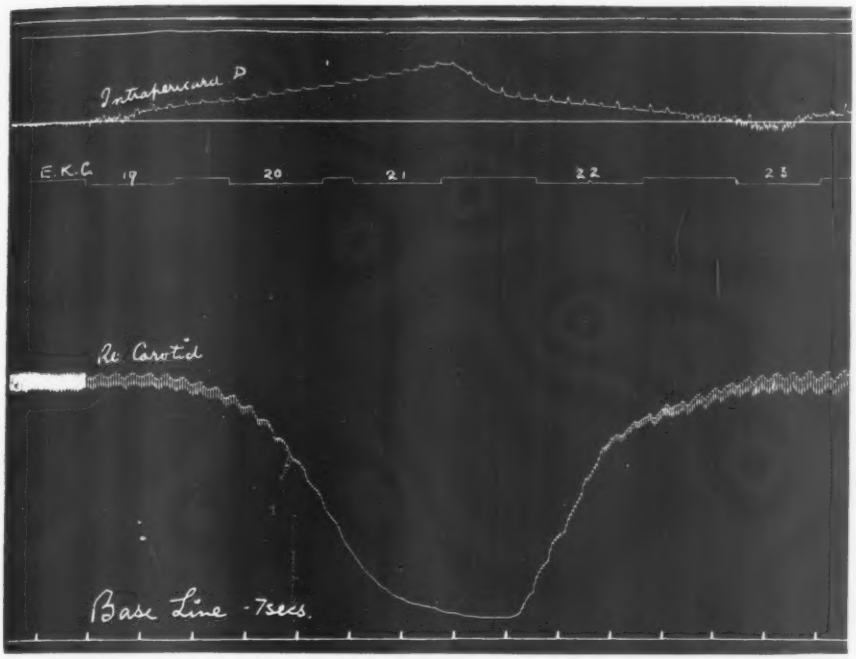

Fig. 1.—The influence of intrapericardial pressure upon blood pressure.

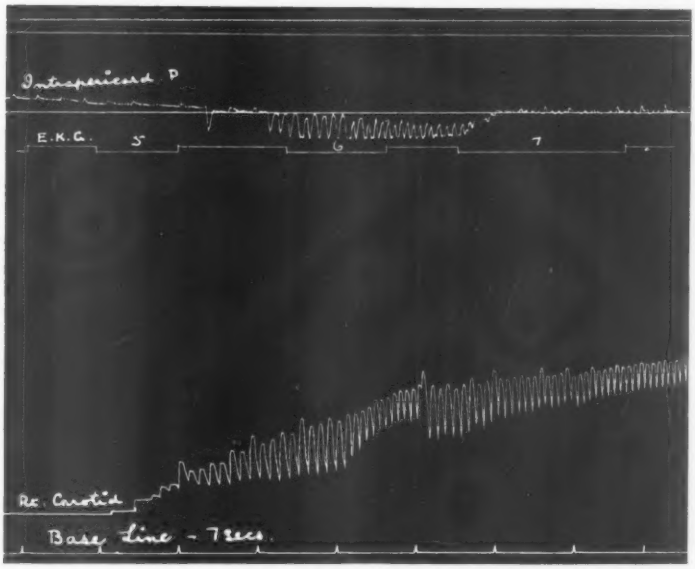

Fig. 2.—Recovery from increased intrapericardial pressure. Note the large pulsations in the carotid artery.

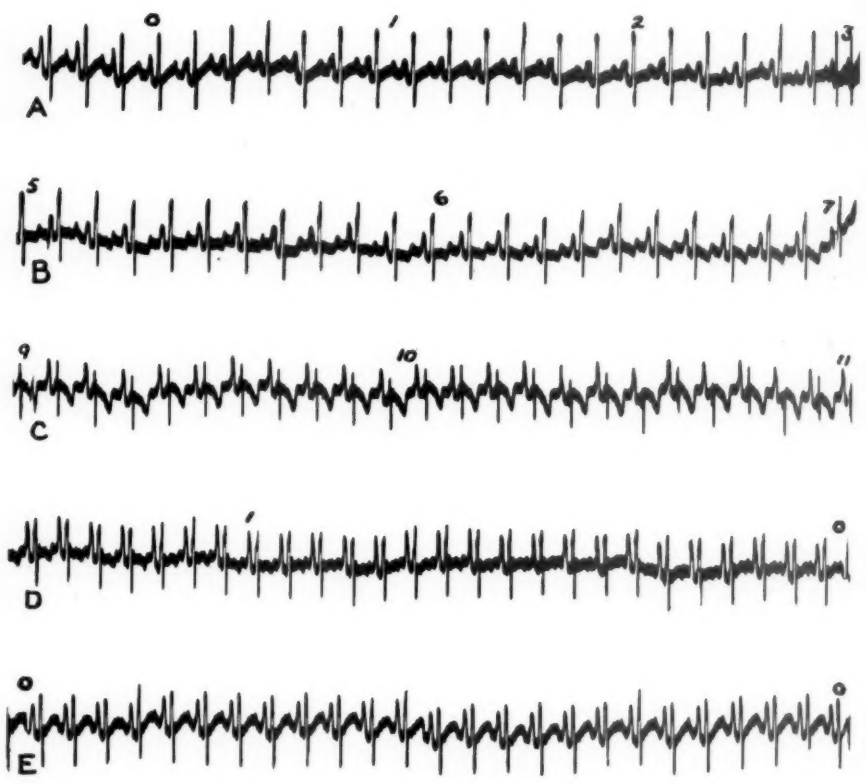

Fig. 3.—The electrocardiogram during changes in intrapericardial pressure. *A*, *B*, *C* during increasing pressure. Note the gradual inversion of the T-waves. *D*, *E* during recovery on release of pressure. The numerals indicate the intrapericardial pressure in millimeters Hg.

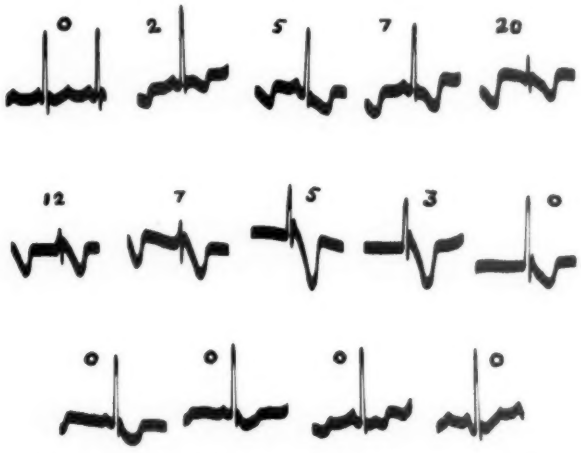

Fig. 4.—Striking examples of QRS segments and T-waves during changes in intrapericardial pressure. The numerals indicate pressure in millimeters Hg. The last 8 examples were obtained during times marked 5 to 7 on Fig. 2.

The intrapericardial pressure necessary to produce a minimal blood pressure varied in our experiments from 15 to 19 mm. Hg. Cohnheim does not mention the specific gravity of the oil used in his pericardial manometer, but assuming it to have a value of 0.90 to 0.95 the pressure of 240 mm. oil which he usually found adequate to reduce the pulmonary arterial pressure to a minimum would correspond to from 15.8 to 17.0 mm. Hg.

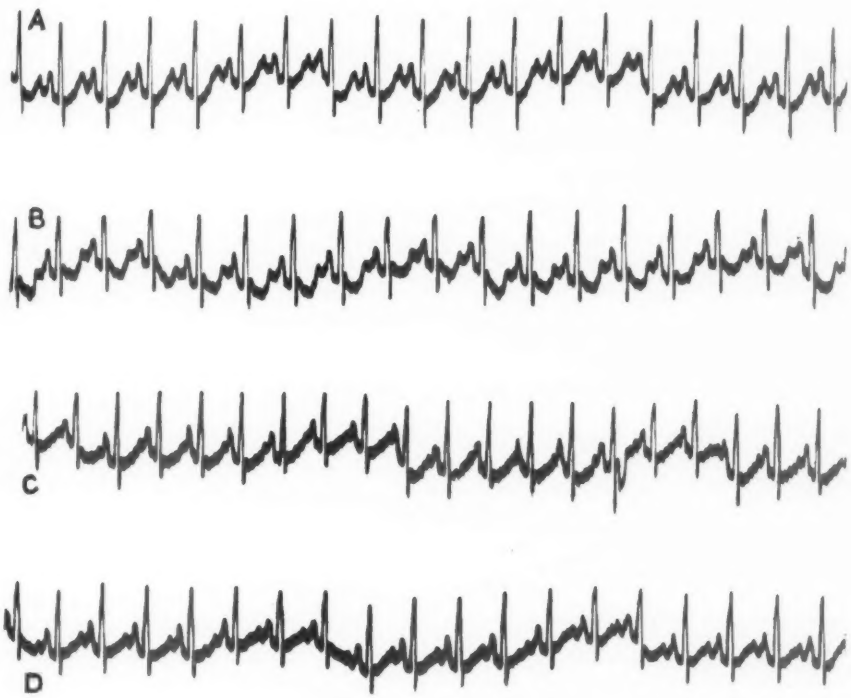

Fig. 5.—The influence of adrenalin on the electrocardiogram in increased intrapericardial pressure. *A* is a control. *B* shows inversion of T-waves by a pressure of 4 mm. Hg in the pericardial sac. *C* is a record taken six seconds after injecting 1 c.c. of 1:10,000 adrenalin solution into the femoral vein. The arterial pressure was beginning to rise at the commencement of this record. Note the restoration to positive T-waves. *D* was taken thirty-five seconds after the injection of adrenalin. The pressor effect of the drug had worn off.

The changes in the electrocardiogram during increased intrapericardial pressure and during recovery on release of pressure are shown in Figs. 3 and 4. Fig. 3 is an almost continuous record of one experiment. Fig. 4 shows stages in production of coronary T-waves. This figure is constructed from records of the same experiment as is shown partly in Fig. 2, the last 8 tracings being from electrocardiographic records taken during times marked 5, 6 and 7 on that figure.

These changes in the electrocardiogram are similar to those recorded by Scott, Feil and Katz. The most marked features are the diminution in voltage of the QRS segments and the gradual inversion of the T-wave with production of the coronary type. During recovery on release of

pressure there was a gradual return to normal, but usually in the early stages of recovery the QRS segments were of greater than normal voltage, and there was an increase in ventricular rate.

By correlation of electrocardiographic with kymograph tracings it was found that a definite change in the T-wave toward negativity was always produced by an intrapericardial pressure of from 3 to 8 mm. Hg.

It appeared of interest to examine the influence upon the electrocardiogram of pressor drugs injected while the intrapericardial pressure was increased. Adrenalin (1 c.c. of 1 in 10,000 solution) showed the typical blood pressure rise if the intrapericardial pressure were not too high. There was sometimes, but not always, a corresponding rise in the intrapericardial pressure. Adrenalin restored the T-waves to their normal positivity, this effect commencing even before the rise in arterial pressure and continuing after the pressor effect of the drug had disappeared (Fig. 5). This result was still shown when the rise in arterial pressure caused an increase in intrapericardial pressure. If, however, the pressure in the pericardial sac were sufficiently high at the time of injection of adrenalin, there was no rise in blood pressure and the changes in T-waves were absent. But the blood pressure rose in the usual fashion immediately the intrapericardial pressure was released. If the pressure in the pericardial sac were sufficient to produce a minimal arterial pressure at the time of injection of adrenalin, then, on release of this intrapericardial pressure, the heart beat very irregularly. The intravenous injection of 2 c.c. of a 0.5 per cent solution of ephedrine sulphate while the intrapericardial pressure was raised produced the same changes as did adrenalin. And once more the pressor effect was absent if the intrapericardial pressure were too high and appeared immediately this pressure was released. A large pericardial effusion obviously greatly hinders circulation.

In applying to clinical studies the results of such experiments as are recorded above, it is important to consider the factor of time. In the laboratory one can only observe the influence of acute pericardial effusions upon blood pressure and the electrocardiogram. In clinical practice one meets more often with effusions slowly accumulating. Even in the laboratory one observes a gradual relaxation of the pericardium sufficient to compensate for the acutely produced intrapericardial pressure. This is especially the case when the pressure produced is of that order just sufficient to cause a reversal of T-waves, viz., a pressure of 3 to 8 mm. Hg. Unless such a pressure be maintained by increasing the volume of the injected fluid, it will be spontaneously and rather rapidly diminished by stretching of the pericardium and the electrocardiogram will revert to normal. It is understandable, therefore, that single, isolated observations of blood pressure and the electrocardiogram in clinical cases will often fail to show the abnormalities which have been produced in our experiments. When such abnormalities are observed clinically, then one may conclude either that the pericardial effusion is of very recent forma-

tion or that it is of such a volume as to have extended to the limit the power of relaxation of the pericardium and this volume of effusion must be greater than would be necessary, in the laboratory, to produce equivalent changes in blood pressure and cardiac activity.

Pyopericardium is uncommon clinically. Yet the rapid accumulation of a purulent exudate in the pericardium may reproduce the conditions of our experiments, as in the case reported by Harvey and Scott.[5] The coronary type of T-wave, associated with pain, fever, lowered blood pressure and leucocytosis, is most often seen in cases of coronary occlusion. Unless the physical signs of pericardial effusion are clear or suggestive, the differential diagnosis may be difficult or impossible. X-ray films of the chest may aid in the differentiation.

SUMMARY

1. The increase in intrapericardial pressure caused by an acute pericardial effusion produces a fall in arterial blood pressure.

2. If the intrapericardial pressure be increased by stages, there is a definite blood pressure established for each stage. This blood pressure is maintained for a minute or more, but there finally occurs a stretching of the pericardium which tends to restore normal conditions.

3. If the intrapericardial pressure be slowly and continuously increased, there is a definite mathematical relationship between the pressure in the pericardial sac and the arterial blood pressure.

4. During an increase in the intrapericardial pressure there occur changes in the electrocardiogram, as described by Katz, Feil and Scott, consisting chiefly of a gradual diminution in the voltage of the QRS segments and development of and increase in negativity of the T-waves, this latter change extending even to the production of T-waves of the coronary type.

5. An intrapericardial pressure of 3 to 8 mm. Hg is sufficient to produce an obviously negative T-wave.

6. When the intrapericardial pressure is sufficiently high to produce negative T-waves, the intravenous injection of adrenalin or ephedrine sulphate solutions will restore the electrocardiogram to normal. This change commences before the pressor effect of the drugs is evident on a carotid pressure tracing and persists after that effect has worn off. It is shown also even if the rise in arterial pressure is accompanied by a rise in intrapericardial pressure.

7. If the intrapericardial pressure be sufficiently high, the circulation is hindered to such an extent that neither adrenalin nor ephedrine will cause a rise in arterial pressure. But this rise occurs immediately on rerise in intrapericardial pressure.

8. The negative T-wave may resemble in all respects that seen in coronary occlusion. Since the rapid accumulation of a purulent exudate in the pericardium may be accompanied by intense pain, lowering of the

blood pressure, fever and leucocytosis, suggesting coronary occlusion, the presence of a coronary T-wave in such a case may offer great diagnostic difficulties.

The authors wish to express their sincere thanks to Dr. R. S. Morris for his continuous and encouraging interest in this work.

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PARTIAL BUNDLE-BRANCH BLOCK*†

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ATTENTION has been called recently to the occurrence of transient bundle-branch block of various types,[1-14] which usually takes the form of a 2:1 partial bundle-branch block. In the cases reported there have been included cases of alternation between right and left bundle-branch block. Transient bundle-branch block has been assumed to occur as the result of functional disturbances in the conduction paths, the cause of which has been ascribed to various factors. An unusual case of partial bundle-branch block of one bundle associated with bundle-branch block of the opposite bundle (at times leading to alternation) and incomplete A-V block of first and second degrees, is presented because it throws light on a mechanism which can produce partial bundle-branch block.

CASE REPORT (SUMMARY)

Dinah P., housewife, aged sixty-five years, was admitted to the hospital on Jan. 3, 1931, complaining of an attack of dizziness and faintness. She noticed these attacks for the first time about three weeks previously. These attacks came on at work with only moderate exertion, lasted several minutes, and became as frequent as three a day before admission.

She had been under medical management for peptic ulcer for the past five years; otherwise her past history is irrelevant.

On physical examination the patient appeared aged, undernourished and was lying comfortably in bed. She had an arcus senilis and bilateral nuclear cataracts. The lungs were clear throughout. The heart was but slightly enlarged to the left, and its sounds were distant and muffled. A presystolic gallop rhythm was present. The aortic second sound was accentuated and louder than the pulmonic second. The pulse rate was 64 and grossly irregular.

The patient was kept in the hospital until Feb. 27, 1931, during which time various therapeutic procedures were tried, as follows:

1. Oxygen tent, Jan. 6 to Jan. 12.
2. Atropine sulphate gr. 1/50 hypodermically (a) Jan. 23, (b) Feb. 4.
3. Camphor in oil hypodermically, 1 c.c., Jan. 28.
4. Caffeine benzoate hypodermically, 7½ gr., Feb. 1.
5. Adrenalin, 1 c.c. of 1/1000 solution hypodermically (a) Jan. 27 (b) Feb. 25.
6. Glucose, 50 c.c. of a 50 per cent solution intravenously, and insulin, 25 units intramuscularly (a) Feb. 18, glucose given first; (b) Feb. 3, insulin given first.
7. High carbohydrate diet, as tolerated, consisting of protein 50 gm., fats 80 gm., carbohydrates 250 gm. Feb. 7 to Feb. 27.

Laboratory Data.—Wassermann reaction was negative. Blood sugar ranged from 76 to 86 mg.; blood nonprotein nitrogen ranged from 38 to 58; blood creatinine ranged from 1.7 to 2.0; and blood cholesterol ranged from 227 to 231. On Jan. 5,

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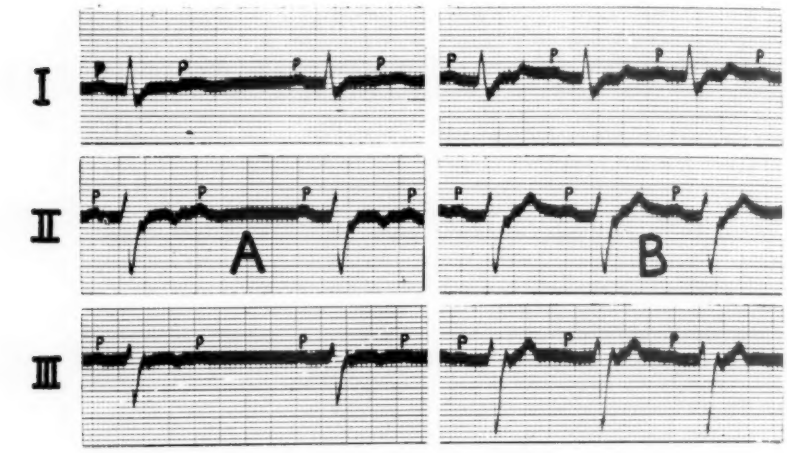
†Aided by the Emil and Fanny Wedeles Fund of the Michael Reese Hospital for the Study of Diseases of the Heart and Circulation.

the red blood cells were 4,240,000, hemoglobin 70 per cent; her blood pressure varied from 130/65 to 158/65; and the urine and stools were negative on repeated examination.

The diagnosis on discharge was arteriosclerotic heart disease, Stokes-Adams syndrome and chronic atrophic emphysema.

DISCUSSION

The various therapeutic regimes were without apparent effect on the cardiac mechanism except in the case of atropine and adrenalin. Atropine produced an acceleration in sinus rate and adrenalin tended



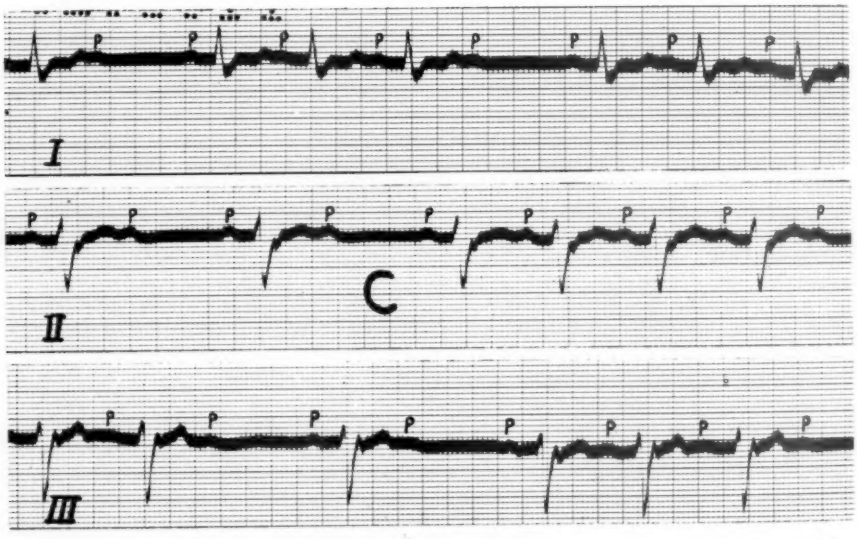


Fig. 1.—Shows the effect of adrenalin on the A-V block. Segment *A*, taken before adrenalin was given, shows 2:1 A-V block with a P-R interval in the conducted beat prolonged and a single type of bundle-branch block, the dominant type. Segments *B* and *C* were taken after the administration of 1 c.c. of 1/1000 adrenalin hypodermically. In segment *B* there is 1:1 conduction with prolonged P-R interval. Note the large, upright T-waves in all leads. In segment *C* there are occasional dropped beats and typical Wenckebach periods. Note that the T-waves are smaller in this segment. In this figure the P-waves are labelled.

to improve the A-V conduction and altered the ventricular deflections by increasing the amplitude of the T-wave. In Fig. 1*A* is shown the change from a 2:1 A-V block before adrenalin (segment *A*) with P-R of 0.26 sec. to a 1:1 conduction with P-R of 0.28 sec. in segment *B* during adrenalin; after the maximum effect of adrenalin had worn off there appeared frequent dropped beats and Wenchebach periods (segment *C*). These changes in A-V block occurred without any alteration in sinus rate. The intraventricular block in this illustration is of the predominant type seen in this patient and according to classical terminology would be called a right bundle-branch block.* The changes in T-wave following adrenalin are clearly seen.

TABLE I

A-V BLOCK	BUNDLE-BRANCH BLOCK (CLASSICAL TERMINOLOGY)	SINUS RATE	AVERAGE
Prolonged P-R	right	54, 55, 56, 56, 58, 58 60, 62, 62, 65*, 65*, 67* 71*, 71*, 72*, 72*	63 (57)†
Prolonged P-R, occasional dropped beats	right	68, 68, 68 72, 73, 75	71
Prolonged P-R, dropped beats	right and left‡	55 60, 62, 66, 68 72, 75, 75*, 77	69
Prolonged P-R, dropped beats	right and left§	60, 62, 67, 68, 68, 68 72, 75, 75, 75, 75, 79, 79, 79 81, 83, 88	74
2:1	right	72, 75, 76, 79 81, 86 94 107, 107	84

*During adrenalin experiment.
†Average of rate omitting adrenalin experiment.
‡"Right" type of complexes occur in some of conducted impulses without previous beat being dropped.
§"Right" type of complexes occur only after previous dropped beat.

An analysis of the 61 sets of curves taken on this patient showed that, considering the variety of conditions under which these records were taken, a correlation could be made between the mechanism and the sinus rate. There were of course other influences besides heart rate which affected the conduction of the impulse through the ventricles as the experiment with adrenalin cited above demonstrated. The presence of a sinus arrhythmia and occasional nodal extrasystoles (viz., Lead III, Fig. 2*B*) further complicated the situation. The data of this correlation are assembled in Table I. At the slower and the faster rates only the so-called right bundle-branch block was present, in the former with 1:1 and in the latter with 2:1 A-V conduction. In the intermediate range of sinus rates the block was less regular, Wenckebach periods occurred and there were frequent dropped beats. In this sinus rate range an

*The classical terminology will be followed in this report.

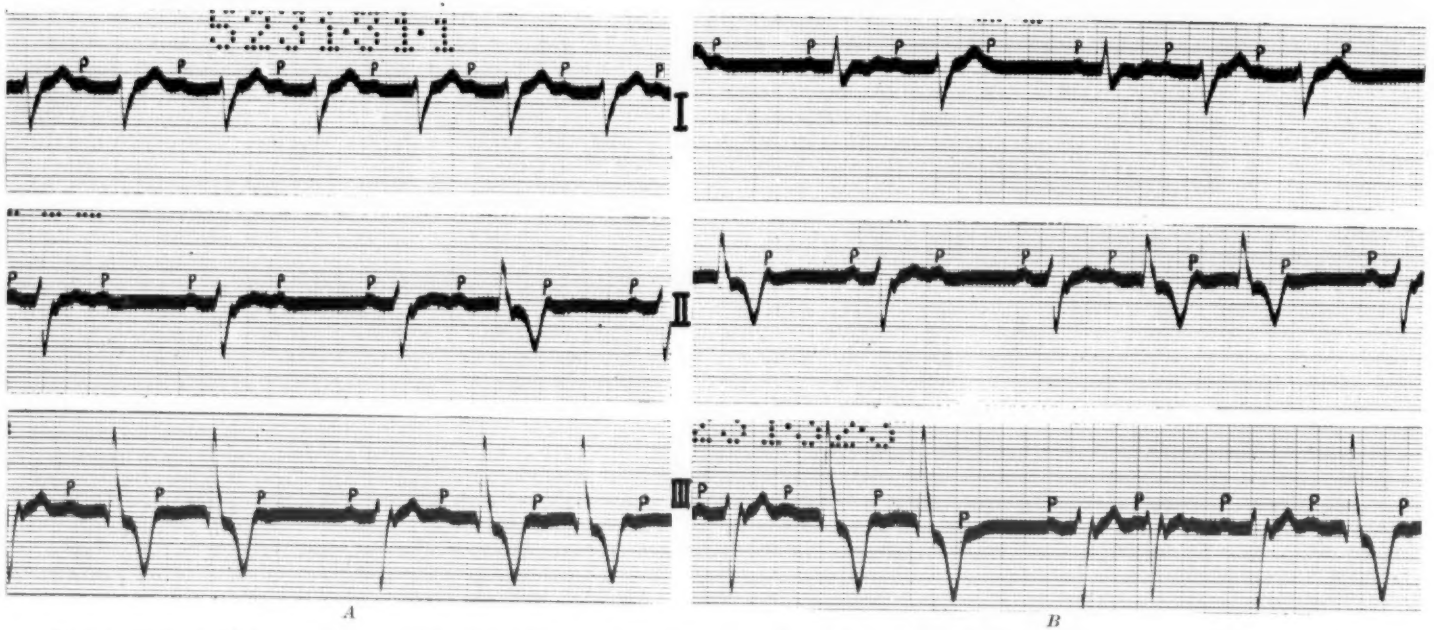


Fig. 2.—*A*, In Lead I is shown 1:1 conduction with prolonged P-R intervals and a persistent bundle-branch block of the uncommon type in this patient. In Lead II there are frequent dropped beats giving rise to 2:1 and 3:2 block. The second ventricular complex of the 3:2 block is of the type shown in Lead I; the rest are of the opposite type. In Lead III all but two are of the same type of bundle-branch block as in Lead I.

In *B* the presence of a partial bundle-branch block is shown. The contrast in configuration of the two types of ventricular complexes is clearly seen. In Lead III there is a nodal extrasystole the ventricular complex of which has the configuration of the dominant type of bundle-branch block. The P-waves in this illustration are labelled.

interesting phenomenon appeared, viz., transient block of the other bundle branch (the left). The change in block from right to left occurred at times for a number of successive beats as in Lead I of Fig. 2*A*, on the one hand, and for an occasional beat, as in Lead II of Fig. 2*A*, on the other. Other combinations were seen, viz., 4:3 block with the right type after the blocked auricular impulse, the left in the others, as in Lead III of Fig. 2*A*; and 3:2 block as in Fig. 3*A* with an alternation of the type of bundle-branch block between right and left, the former occurring after the blocked auricular impulse. Irregular types such as shown in Fig. 2*B* and 3*B* were also observed often. Wenckebach periods with progressive lengthening of P-R occurred when the transient left bundle-branch block was present, just as was the case when the right bundle-branch block was present (compare Lead III of Fig. 2*A*, all leads of Fig. 2*B*, and Fig. 3*B* with all leads of Fig. 1*B*). In fact the progressive lengthening of P-R was equivalent in the two types of bundle-branch block as can be seen by comparing the P-R intervals of the

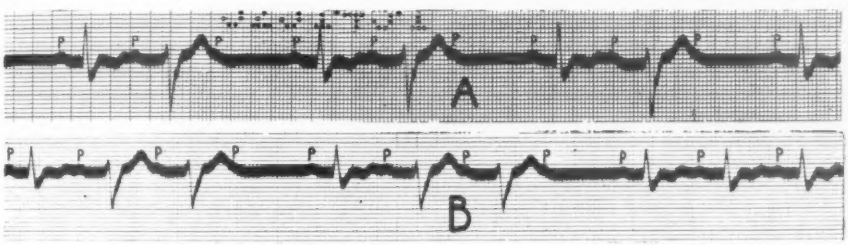


Fig. 3.—Segment *A* shows an alternans between the two types of bundle-branch block, associated with a 3:2 A-V block. Segment *B* shows in a single lead the appearance of 1:1 conduction of the two types of bundle-branch block. The P-waves in this illustration are so labelled.

last three ventricular complexes of Fig. 3*B* with the first three or middle three.

There can be no question that the severity of the A-V block was dependent on the sinus rate; the block increasing, with some variation, from a simple prolongation of the P-R interval to a 2:1 conduction. The administration of adrenalin was the only procedure with noticeably altered this relationship by improving A-V conduction without changing the sinus rate. The other therapeutic procedures either had no apparent effect or acted by changing the sinus rate, as in the case of atropine.

The occurrence of a transient left bundle-branch block was limited to the intermediate sinus rate range (see Table I). In no instance did this transient left block occur after a dropped beat. In other words, it always occurred after at least one previous conducted impulse. The following explanation is offered to account for this peculiar phenomenon:

There is in this case a permanent organic damage in the right bundle branch and in the A-V junctional pathway. The block in this bundle branch is unaffected by changes in heart rate in the range studied; that of the A-V junctional tissue varies with the sinus rate. The block

in the left bundle is more extensive than in the right but is not permanent. If sufficient time is allowed for recovery after the passage of an impulse, before the following impulse reaches it, the impulse will pass without any appreciable delay. If, however, the second impulse comes earlier, it is completely blocked; that is to say, it reaches the left ventricle by a circuitous route. Of course, the impulse is delayed to both ventricles under these circumstances but reaches the right sooner than the left. Since the QRS complex is not registered until some of the ventricle is activated, the delay of the impulse in reaching the right ventricle can only prolong the P-R slightly; and since the right is activated before the left ventricle, the complex will take on the appearance of left bundle-branch block.

SUMMARY

A case is reported in which there is a combination of a region of permanent and complete block of one bundle branch associated with less complete but more extensive block in the other bundle, and complicated by the presence of incomplete block of the A-V junction. The presence of this combination with changing ventricular rate caused the appearance of transient bundle-branch block of one type to be superimposed on that of the opposite type, with, at times, an alternation between the two.

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POSSIBLE INTRANODAL BLOCK. A REPORT OF CASES*

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THIS report is concerned with electrocardiograms from three cases showing rhythmic deflections at a regular rate, unrelated to the normal auriculo-ventricular complexes.

In one case these deflections are identical in form with the P-waves of the auriculo-ventricular complexes. The latter complexes have normal P-R intervals. The electrocardiogram from this case gives the impression of a double auricular rhythm, that is, of two sets of auricular contractions, both rhythmical, but unrelated to each other. The extra rhythm in the other two cases does not appear so evidently auricular in origin, but some features are suggestive.

CASE REPORTS

CASE 1. J. K., Unit No. 25076, a white male twenty-two years old, was admitted to the hospital because of acute rheumatic fever. The onset was sudden with stiffness, pain and swelling of both ankles together with fever and sweating. The patient had noted these symptoms six weeks prior to entrance upon waking from a nap taken in his automobile on a rainy day. The symptoms had diminished somewhat at the time of entrance. Anorexia had been present for the first three weeks, and during the six weeks the patient had lost twenty pounds in weight. Otherwise the history was insignificant.

Physical examination showed the pharynx to be injected, the left tonsil large, cryptic and moderately injected. The anterior cervical chain of glands was palpable on both sides, but the glands were larger on the left (almond size). Swelling and redness in the region of the malleoli were present in both ankles. The skin was damp and cold. Otherwise the examination was negative.

The temperature was 101.6° F. rectally, the pulse rate was 90 per minute. The leucocyte count was 16,000 per c.mm. The red blood count and hemoglobin were normal. The Wassermann and Kahn tests were negative. The urine was normal.

Within six days after admission the temperature was normal, the excessive perspiration had stopped, and the swelling and pain in the joints were less, although during that time he had complained of pain and stiffness in the right shoulder and fingers, and in the lumbar region at times. On two or three occasions a soft apical systolic murmur was heard, but this was not constant. Electrocardiograms were taken on the tenth and eleventh days of hospitalization. On the fourteenth day the symptoms had disappeared and the patient left the hospital against advice.

In Lead I of the first electrocardiogram, (Fig. 1*A*) the peculiar appearance of two independent sets of auricular waves is seen. This finding was not present in the tracing taken the following day.

It is quite evident that in this case there is no noticeable difference in the form of the auricular waves preceding the ventricular complexes and the waves of the other set. This other set is rhythmic, we think, although

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some of the waves are not evident because of coincident ventricular complexes. The intervals between the waves vary from 0.42 to 0.56 seconds with an average rate of 113 per minute. The auriculo-ventricular complexes occur at an average rate of 85 per minute. The curve shows a slight sinus arrhythmia, and both sets of waves vary practically together but not to the same extent. This relation, together with the similarity in form of the regular auricular waves and the waves of the other set, makes it probable that they are both cardiac in origin.

CASE 2. L. M., Unit No. 382, a Jewish girl nine years old, has been under observation from time to time for the past three years. Prior to admission here the only past history of importance was scarlatina at the age of six, uncomplicated. There is a family history of syphilis.

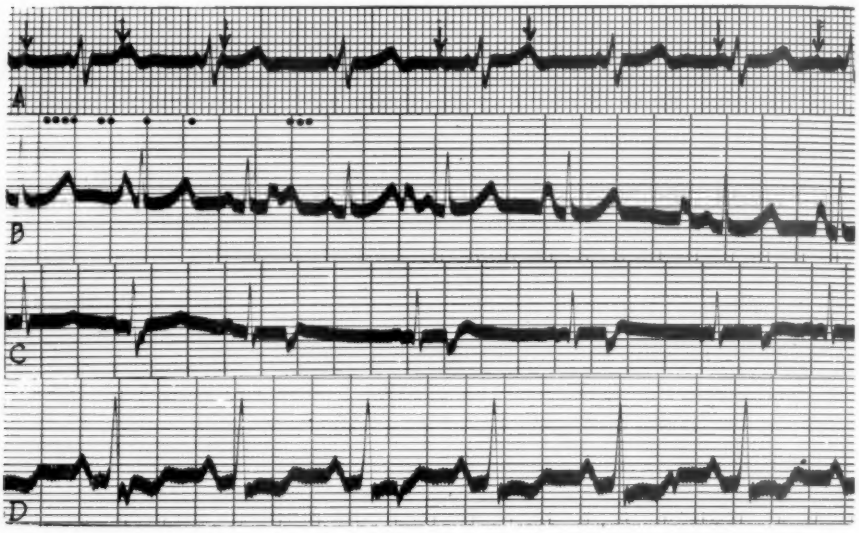

Fig. 1.—Short sections of electrocardiograms from the cases with abnormal rhythmic deflections. *A*, Lead I from Case 1. The abnormal waves are indicated by arrows. *B*, Lead I from Case 2. *C*, Lead III from Case 2. *D*, Lead II from Case 3.

Although she was examined several times during these three years, nothing referable to the cardiovascular system worthy of note was discovered until June of 1931, with the possible exception of an attack of acute tonsillitis, for which tonsillectomy was finally performed in June of 1930.

In June of 1931 she was hospitalized because of a gastrointestinal upset with slight temperature, nausea, vomiting and generalized abdominal pain which persisted for five or six days. The leucocyte count rose as high as 15,500, with 75 per cent polymorphonuclears. The gastrointestinal symptoms, temperature and leucocytosis subsided entirely, but shortly before discharge a duplication of the first heart sound at the apex was noted. The blood pressure was 106 mm. of mercury systolic and 76 mm. diastolic in the sitting position. In the supine position the systolic pressure was 102 mm., and the diastolic pressure could not be read, the sharp sounds persisting to the zero mark. Marked sinus arrhythmia was present.

An electrocardiogram was taken, Leads I and III of which are shown in Fig. 1 *B* and *C*. From that time until the present the patient has been followed and six electrocardiograms have been taken, without any reappearance of the peculiar de-

flections of the first tracing. The heart findings remain the same except that recently there has been a loud musical murmur, best heard in the second interspace to the right of the sternum, at times to-and-fro, and at times limited to diastole. This murmur is present only when the patient is in the upright position, disappearing entirely when she lies down. The blood pressure readings remain the same.

The electrocardiogram from this patient shows deflections in Leads I and III similar to those in Lead I of the first case in that they vary in rate with the variations in rate of the normal complexes due to sinus arrhythmia but not to the same extent. The form of the waves, however, is markedly different from that of the P-waves preceding the ventricular complexes.

CASE 3. J. C., Unit No. 31513, a white male twenty-five years old, with chronic glomerulonephritis, hypertension, hypertensive neuroretinitis and right facial paresis showed the electrocardiographic manifestations in question (Fig. 1 *D*) about a week before his death while he was suffering from myocardial insufficiency and uremia, although several previous and one subsequent tracing furnished no further evidence of this abnormality.

In this case Lead II shows rhythmic deflections at a slow rate, which is slightly variable. The interval varies from 1.25 to 1.45 seconds with an average rate of 42 per minute. The rate of the usual auriculo-ventricular complexes is absolutely regular at 92 per minute.

The identity of these rhythmic deflections as evidences of auricular activity in Cases 2 and 3 is not so apparent as in Case 1.

DISCUSSION

Without the first case the other two might not be of sufficient interest to merit special consideration, although it would be difficult to explain the abnormal waves as artefacts. Artefacts might possibly occur rhythmically, as the result of the rhythmic contraction of some extracardiac muscle, but it is difficult to conceive of an extracardiac rhythm variable with the variability in heart rate due to sinus arrhythmia. We believe that these waves are manifestations of cardiac activity, at least in Case 1.

Case 1 has one peculiarity, not shared by the others, that makes it seem even more likely that the waves are of auricular origin; namely, that the abnormal waves are identical in form with the P-waves of the normal complexes. In this case, as in the others, the waves are rhythmic and the rhythm varies with the sinus arrhythmia of the heart.

Fig. 2 *A* shows the rates of the two rhythms plotted from Case 1. It will be noted that in general the intervals between the normal complexes and the intervals between the abnormal waves lengthen and shorten simultaneously. Fig. 2 *B* and *C*, shows similar graphs constructed from the curves from Case 2. No graph from Case 3 is shown because, although the rhythm of the abnormal waves is slightly variable suggesting a sinus arrhythmia, the rate of the normal complexes is absolutely regular. This makes it impossible to determine whether the variations in rate are synchronous with respiration or not.

The abnormal waves occur in very close proximity to normal auricular waves in some instances, and very soon after the ventricular complexes in others (see the second ventricular deflection in Fig. 1 *A*). They are present so soon after the ventricular complexes that the muscle whose stimulation gave rise to the ventricular complex must still be in a refractory phase. Since the QRS complexes are normal in appearance, it is extremely likely that all the ventricular muscle is stimulated and contracts at the time of the ventricular complex. If this is true the abnormal waves cannot be the result of ventricular activity. The same argument applies in the case of the auricular waves except that the auricular complex is not so characteristic as the QRST, and it would be difficult to determine whether all the auricular muscle was stimulated at the time

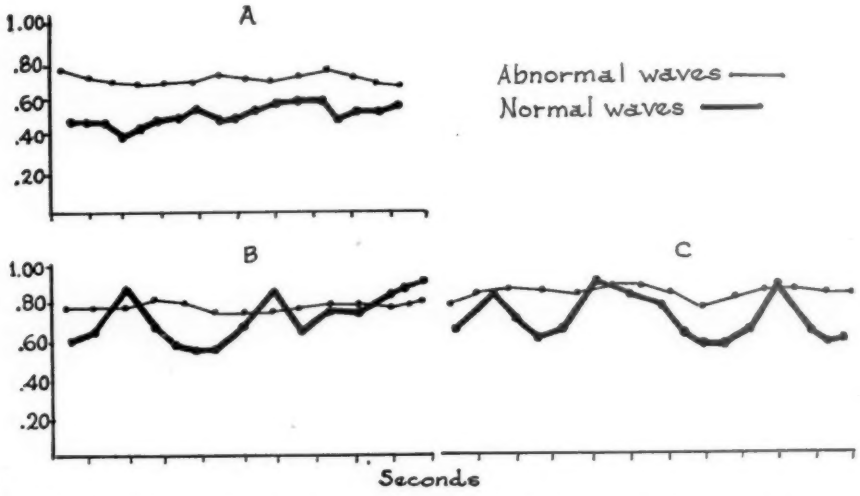

Fig. 2.—Graphs constructed from the electrocardiograms of Cases 1 and 2. The vertical distance represents the time in seconds between the P-waves of the normal complexes (heavy line) and between the abnormal waves (light line). The horizontal distance represents absolute time in seconds. *A*, Lead I from Case 1. *B*, Lead I from Case 2. *C*, Lead III from Case 2.

of the P-wave. It is possible, therefore, that the normal P-waves and the abnormal waves are the electrocardiographic expressions of the activity of separate portions of the auricular muscle.

This view is supported further by the fact that the abnormal waves are subject to the influences that cause sinus arrhythmia in the rate of the normal cycles. These influences are neurogenic and are carried by the extrinsic cardiac nerves. It may be inferred that their effect is manifested chiefly in the region of the sinus node, because in cases of auriculoventricular nodal rhythm respiratory arrhythmia is rarely if ever seen. While this inference is open to some question, it makes it possible, or even probable, that the point of origin of the stimulus giving rise to the abnormal waves is close to, or part of the sino-auricular node. This suggests the possibility of a condition of block within the sino-auricular

node with separate portions of the node originating different sets of P-waves.

We have found only one case similar to these in the literature. Schrumpf,[1] in 1920 reported the case of a man, thirty-seven years old, with rheumatic heart disease, mitral regurgitation, aortic insufficiency, and myocardial insufficiency, who, in a state of digitalis intoxication showed a tracing similar in many respects to the tracing from our Case 1. In his patient the apparent double auricular rhythm persisted for three days and disappeared as the digitalis intoxication subsided. He believed that because in many instances the P-waves and the abnormal waves were very close, they could not arise from the same nodal tissue, because of the refractory period of nodal tissue.

He pointed out the desirability of demonstrating a double set of auricular waves in the venous pulse tracing, corresponding to the two sets of apparent P-waves in the electrocardiogram. This could not be done in his case because dyspnea made it impossible to take venous pulse tracings. To support his opinion that the two auricles contracted separately he presented the synchronized electrocardiographic and venous pulse tracings from a patient with broad notched P-waves in which he found a double auricular wave in the venous pulse tracing corresponding to the two peaks of the P-wave. This tracing is suggestive but not entirely convincing. He concluded that the best explanation of the curve was that the Keith-Flack node was of dual character, that its two parts were associated with the two sides of the heart, that the two parts were completely dissociated, and that one auricle followed each part of the node.

In explaining the fact that the ventricles always followed the same set of auricular impulses he considered it probable that one set of impulses was relatively weak and that a condition comparable to auriculo-ventricular block existed between the portion of the node giving rise to the weak set of impulses and the auriculo-ventricular node, while the impulses giving rise to the other set of P-waves were stronger and the ventricles followed these as usual.

Schrumpf's article is a case report and is not supported by experimental work. It is of interest because of the similarity of the electrocardiograms from his case and with those from ours and because of his suggestion of the duality of the sino-auricular node.

Pace[2] described the gross and microscopic form of the sinus node in the heart of a sheep which he studied. In addition to the portion of the node extending from above downward and to the right in the region of the cavo-auricular sulcus, he found a prolongation extending from the upper end downward and to the left in the cavo-auricular cone, terminating in the interauricular septum. He did not generalize regarding these findings but thought it likely that they were an anomaly of the heart of the animal he studied.

Bruni,[3] at the same time as Pace, published a detailed anatomical study of the region of the sino-auricular node. He studied the region in embryos of the sheep and ox, and selected the 20 cm. ox embryo for a detailed description because at this stage of development the structures were particularly well shown, and the node could be reconstructed in detail accurately. He was able, however, to show the same general structure in hearts up to term.

In the 20 cm. fetus the node consisted of a body in the form of a quadrilateral plate in the region of the terminal sulcus on the external surface of the anterior part of the superior vena cava, coming in contact with the pericardium. This plate extended into the sinus portion of the right auricle. Several branches of nodal tissue were described extending from this plate, one from each anterior corner, left and right, the one on the left terminating in the interatrial sulcus. These were short and conical in form. Another, longer than these, extended up the right anterior wall of the superior vena cava. Two prolongations extending obliquely in a caudal and ventral direction are of particular interest. One extended in the wall of the right auricle from the right side of the central plate toward the inferior vena cava but did not reach it. The other extended from the left side of the central plate into and through the interauricular septum. No direct communication between either of these branches and the node of Tawara was demonstrated.

The studies of Pace and Bruni have this in common: they both found extensions of sinus nodal tissue into the interauricular septum.

Géraudel[4] makes reference to this work and to an anatomical study by Segré, who, in serial sections from four human hearts, two in fetuses, one in a newborn, and one in a young person, demonstrated a horseshoe shaped mass of nodal tissue in the superior vena cava. The left branch of this nodal tissue extended into the interauricular septum. This left branch he termed the node of Pace and Bruni. Géraudel was unable to demonstrate this left sided nodal tissue in adult hearts but thinks that the work of the several men on this subject deserves further consideration.

Roversi[5] reviewed the findings of Pace and Bruni in animals and reported experimental work of his own on dogs in support of their work. He believed that there were two masses of sino-auricular nodal tissue connected by specific conduction tissue. He also believed that the original node of Keith and Flack initiated the impulse, which was transmitted to the node of Pace and Bruni and thence to the ventricles. According to his conclusions the latter node could assume the functions of pace-maker in the event the node of Keith and Flack was destroyed. He was able to demonstrate to his own satisfaction, experimentally, that if the conducting tissue between the nodes was destroyed two P-waves resulted electrocardiographically. He interprets Schrumpf's curve ac-

cording to his work to indicate a dissociation of the two parts of the sinus node. He agreed with Schrumpf's conclusion that it was probable that each auricle had its separate node and could be stimulated independently.

One weakness of his argument in explaining the double auricular rhythm is the lack of evidence for his contention that the two auricles, as units, can be stimulated to contraction separately. Inasmuch as it is generally believed that an auricular stimulus spreads by way of the ordinary myocardial tissue through both auricles, more evidence than he presents will be necessary to prove the hypothesis.

Borman and Meek,[6] in a study of hearts in dogs in which the sinus node had been destroyed by radon, found that the impulse arose in the region of the coronary sinus. This was in accord with the previous findings of Eyster and Meek. The latter, destroying the node surgically, found a shortened P-R interval when the sino-auricular node was not active. Borman and McMillan,[7] using radon, obtained normal electrocardiograms with the sinus node destroyed. These findings are interesting in connection with a possible double auricular rhythm, if we consider the possibility of a dissociation of the sinus node and the region of the coronary sinus, with both of them giving rise to stimuli at regular but different rates. An added difficulty arises in fitting this conception to our curves, however, in that the two centers are in the same auricle.

In spite of the fact that we do not consider the conception of the dual character of the sino-auricular node adequately proved, we are unable to controvert it, but can only raise questions and objections. It offers an explanation of the curves we have seen which would be difficult to make under other circumstances, although other possibilities must be considered. Two of the chief alternative hypotheses are: first, that the independent waves represent auricular extrasystoles which are not followed by ventricular responses, and second, that the electrocardiographic deflections are due to some extracardiac influence. The first is open to one of the same objections as Roversi's hypothesis, that is, the impulse giving rise to the auricular extrasystole is never propagated to the ventricles. The second seems unlikely, because in the two cases in which there is a sinus arrhythmia in the rhythm of the normal complexes the rate of the abnormal waves is variable to some extent and the variations occur in rather close harmony with the sinus arrhythmia of the normal complexes, though not to the same extent.

The papers of Schrumpf, Pace, Bruni, Roversi and Segré suggest the existence, perhaps as an anomaly, of a potentially double sino-auricular node, the two elements of which may be dissociated under rare conditions by what might be termed an intranodal block. We are unable to come to any conclusions about the mechanism producing these electrocardiographic manifestations. In any event the existence of such curves in the presence of disease processes known to produce defects in intra-

cardiac conduction probably should be regarded as evidence of myocardial change, even in the absence of any other objective sign of the latter.

SUMMARY AND CONCLUSIONS

Some curves are presented showing an unusual transient disturbance of rhythm, associated in all instances with other cardiac signs or symptoms. No adequate explanation for the formation of the curves is given, although possible mechanisms are discussed, among the most likely being intranodal block. When encountered in the course of pathological processes potentially damaging to the conduction mechanism of the heart such as those present in acute rheumatic fever, diphtheria, arteriosclerosis and others, they probably should be regarded as confirmatory, if not conclusive, evidence of such damage.

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INCIDENCE AND DEVELOPMENT OF HYPERTENSION AND HEART DISEASE IN RAILROAD EMPLOYEES*

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IN THE past four years 1,390 physical examinations have been made in the office of the Division Medical Examiner of the Chicago, Burlington, and Quincy Railroad. Of these, 618 were made on prospective new employees, 679 were service reexaminations, while 93 were examinations either for promotion or for increase in relief benefits—a term which really signifies increasing the insurance carried for them by the company, the premiums being deducted from their wages.

While becoming a member of the "relief" is not compulsory, nearly 100 per cent of the employees on our division are members. They must pass as good an examination to be employed as to become a relief member. Besides requiring a physical examination on all prospective new employees, the medical department of the road also has decreed that all old employees having anything to do with the running or maintenance of trains and equipment shall have a physical examination at least every two years. This includes engineers, firemen, conductors, brakemen, flagmen, yardmasters, switchmen, switch tenders, dispatchers, wire chiefs, telegraph operators, crossing gatemen, bridge and building foremen, derrick engineers, and section foremen. If at any time anything develops in the examination of one of these men which would make it expedient to see him more often than every two years, he is notified each year to appear for examination. These examinations are compulsory, *and a man may* be held off duty until examined if he is somewhat dilatory about going to the examiner's office. Usually, in order to help the men to remember, the examinations are arranged to take place in their birth month; and besides that they are notified by mail when their examination is due. Engineers are ordinarily examined every year after the age of fifty and every six months after the age of sixty. If they continue in good physical condition, they are retired and pensioned at seventy.

It is evident that we have had an unusual opportunity for examining a group of men at regular intervals. The development of symptoms and pathological findings has been noted with great interest. There is no question as to when one will again see the patient. We know he will report for examination at a certain time, and if we wish to see him more often, we merely request that he come in every three months or every month if necessary to check up his physical condition. The great majority of the men are interested in keeping fit, and they usually receive and act upon any suggestions for their physical betterment with an admirably cooperative spirit.

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*Read before the Iowa Heart Association, Iowa City, Iowa, January 30, 1932.

The total number of examinations of prospective new employees and of those for promotion or increase in relief insurance was 711; of these 60, or 8.43 per cent, were rejected—practically one out of every twelve. Half of these rejections, or 30 cases, were due to hypertension, heart damage, or a combination of the two. The other 30 men were rejected for the following reasons: diabetes 9, defective vision 6, hernia 3, albuminuria 3, acute urethritis 3, color blindness 2, hypotension 1, defective hearing 1, convulsive syncope 1, and ankylosis of the elbow 1. However, we repeat that one-half of the rejections, or 4.22 per cent, was due to hypertension, heart damage, or both. This is practically one man out of every twenty-five.

The arbitrary upper limits for blood pressure for acceptance as a new employee are 150 mm. systolic and 100 diastolic. Evidence of organic heart disease is sufficient to reject the applicant.

It is interesting to note that only one case of hypertension in association with lues was seen, one case of hypertension and diabetes, and, strangely enough, one case of hypertension and pulmonary tuberculosis.

As mentioned before, 679 service reexaminations have been made. Of course, some men have had several examinations, so that there have not been 679 different individuals. However, we have a list of 74 employees who are now coming in for reexamination at intervals of one year or less. Practically all of these are on the frequent reexamination list because of hypertension, heart disease, or both.

In the past sixteen months there have been four deaths among employees who were being regularly reexamined. Of these only one was unexpected. This was a machinist sixty-six years old who had been in an automobile accident only six and one-half months previous to his death. He was not badly injured but was not passed for work for a period of two months because of some slight evidence of heart weakness. He had a slight hypertension and evidence of arteriosclerosis. He dropped dead in a bank. I have wondered whether accidents, in these older people, in which the injuries are apparently not serious, may not be much more important than we are inclined to think, especially in the upsetting of the circulatory balance. The other three deaths were all due to coronary thrombosis, two associated with marked and one with moderate hypertension. The two men with marked hypertension were forty-three and fifty-one years old, respectively. The man with moderate hypertension was sixty-three years of age. This sixty-three-year-old switch tender had advanced sclerosis. An interesting point was that in the regular reexamination made less than three months prior to his death he complained of pain in the right wrist and hand in the ulnar nerve distribution area. Two days before death he complained rather bitterly about the neuritis in his left elbow and shoulder. He lived only nine hours after the onset of his acute coronary symptoms. The fifty-one-year-old engineeer died suddenly just after getting dressed in the early morning in preparation to drive out the fast mail.

The number of engineers who have died at the throttle is relatively very small. Dr. D. B. Moss, the chief medical officer of the Burlington Railroad, has told me that he knows of only one. The forty-three-year-old fireman died on duty about one hour after acute symptoms appeared. He was luetic, had been reexamined frequently, and the progressive aortic lesion had been watched with the expectation that sudden death might arrive at any time.

Of the 74 whom we are watching closely and examining at more or less frequent intervals, there are six with proved syphilis. These men are watched very closely, as we feel that Dr. Carey F. Coombs is right when he states that aortitis is the inevitable lot of the syphilitic who is inadequately treated in the early stages of the infection. Practically all of the luetic patients that I see at present have been inadequately treated. Incidentally I feel also that Stroud of Philadelphia is right in prophesying that we should be prepared for an increase in the incidence of syphilitic aortitis in the next ten to twenty years as an aftermath of the war. Aortitis seems to resemble tabes and paralysis in the period of the disease in which its appearance occurs; i. e., about twenty to twenty-five years after the primary infection.

Of the six luetic employees we are watching, five are engineers and one is a switchman. The switchman has some perforations of his palate and his uvula is missing, but so far his circulation has been good, with a blood pressure of 144/98 and no sign of heart or aortic damage. Only one of the engineers is in difficulty. He has beginning tabes and one of the most labile types of blood pressure I have ever seen. It has been recorded at varying levels from 114/90 up to 220/130. At present it has rather stabilized itself at about 190/120.

Blood pressure readings are considered important in this group, as any evidence of persistent drop in the diastolic pressure after other signs of aortic involvement have been found must mean aortic valve insufficiency. If the aortic valves become incompetent, then there must be considerable involvement of the mouths of the coronary arteries, with consequent narrowing, and sooner or later thereafter there is noted a lessening of the pulse pressure which indicates ventricular weakness—the next stage in the downward grade.

The remaining 68 are all seen because of heart damage, high blood pressure, or both, with the exception of one who is seen because of a fairly persistent hypotension of about 100 systolic. Of these 67 only three are seen because of the heart alone (all three being rheumatic hearts), 25 are seen only on acount of hypertension, and the remaining 39 have evidence of heart damage and hypertension.

Most of the hypertension cases are of moderate grade, ranging from 160 systolic in the younger men, or 170 in the older men, up to 190. A very few—only three, I believe—are or have been above 190 systolic. One fireman was found to have a pressure of 210/124 at forty years of age. After some infected teeth were removed, the pressure dropped

to 180/114, and after tonsils were removed it was 164/114. The relatively slight decrease in the diastolic pressure is noteworthy.

It has been observed that, among the younger men, many whose blood pressure had been slightly increased showed a return to normal after the removal of abscessed teeth. It is felt that the suggestions which may be made at the time of the regular service reexaminations may be of importance to the employees.

We have been accustomed to change a man from the biannual to the annual examination if his blood pressure has increased 20 points or more since the last examination, or if the systolic blood pressure is 155 or above or if the diastolic blood pressure is 100 or above. He is also advised concerning the eradication of foci of infection, and we find that infected teeth in these employees are very common and also rather difficult to have removed. Our campaign against abscessed teeth is bearing some fruit, however, and we feel that the educational feature of the measure is sinking in.

The progressive features of circulatory degeneration in some of the cases have been particularly engrossing. The progressive steps in this degenerative process might be listed in a general way as follows: 1, gradual increase of hypertension; 2, appearance of an accentuated aortic second sound; 3, appearance of systolic aortic murmur sometimes closely followed by systolic murmur at apex; 4, gradual diminution of the diastolic blood pressure, with appearance of a to-and-fro aortic murmur or sometimes of ventricular weakness without appearance of aortic insufficiency; 5, in many cases circulatory accidents, either of the cerebral or coronary type.

Several men have been retired because of disabilities which have been considered to be too dangerous to the lives either of themselves or of those entrusted to them. Among these are an engineer, retired because of coronary thrombosis, although it was very difficult to keep him in bed even for ten days after his heart accident. He is up and around but has some dyspnea on moderate exertion. Another is a dispatcher who was retired after an acute attack of uremia with convulsions. It was felt that we should not further entrust the direction of the movement of trains to him. One switchman was retired of necessity after a hemiplegia. One engineer was retired because of slight hypertension and sclerosis with paresthesias of one arm and leg. One year later he suffered a hemiplegia and died in a very short time. A conductor was retired because of hypertension and evident heart damage with sclerotic aortitis.

It is to be hoped that all of our private patients may some day see the wisdom of regular physical examinations, for I believe that the early symptoms of many disturbances may be detected, and the process checked, if their physicians are only given a chance to see them at regular intervals.

A CASE OF TUBERCULOUS PERICARDITIS WITH EFFUSION TREATED BY MEANS OF PNEUMOPERICARDIUM*

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THE injection of air into the pericardium as a therapeutic procedure in pericarditis with effusion was first reported by Wenckebach in 1910.[1] In his patient, who had pulmonary tuberculosis as well as a large pericardial effusion, repeated taps without the injection of air failed to prevent the rapid reaccumulation of the fluid, but following the injection of air in amount equal to about half that of the fluid removed, the reaccumulation was much slower and the general condition of the patient improved. Wenckebach followed his case for a year and a half and in that time did nine taps followed by air injections. At the end of this time the patient was greatly improved.

Following this successful case, which was also reported by Geselschap in 1910,[2] Alexander in 1911,[3] Hansen,[4] and Émile-Weil and Loiseleur,[5, 6] in 1916, Meyer[7] in 1918, Martinet[8] in 1921, Castex[9, 10] and Troisier, Jacquelin and Gayet[11] in 1923, Oppenheimer[12] in 1924, Rigler,[13] Rawls,[14] and Lian and Corneau[15] in 1925, Yacoël and Giroux,[16] and Castex, Carelli and Gonzalez[17] in 1926 each reported one case of probable or proved tuberculous pericarditis with effusion treated by tapping followed by injection of air, oxygen or nitrogen. Zuccola[18] reported three cases in 1925, one of rheumatic and two of tuberculous origin.

Without exception all the authors felt that the procedure gave symptomatic relief and slowed up the reaccumulation of the fluid. Of the sixteen patients with tuberculous pericarditis treated by pneumopericardium nine died while under observation, but of these in one only[14] did death seem related to the procedure. This patient died eight hours after the first air injection. Most of the others died of tuberculosis elsewhere in the body, usually pulmonary, after the pericardial symptoms had been largely or partially relieved. The seven patients who did not die under observation were not followed long enough to permit the drawing of definite conclusions, but several of them recovered sufficiently to resume partial activity.

The chief therapeutic advantage of the procedure has been thought to be the prevention of the rapid reaccumulation of fluid following tapping. In addition, it lessens the resistance the heart has to work against by substituting an easily compressed gas for a noncompressible liquid. Several authors have felt that it is possible to prevent the formation of adhesions by keeping the pericardium distended with air. A systematic effort to do so was made by Zuccola.[18] In two cases of tuberculous peri-

*From the Medical Services of the Massachusetts General Hospital.

carditis he injected air several times after the fluid had disappeared, raising the pericardial pressure in the last taps from 5 to 12 and from 10 to 16 cm. of water, respectively. When these patients came to autopsy, several months after the last tap in each case, there were a few pericardial adhesions, especially about the apex of the heart. Zuccola emphasized the importance of injecting enough gas to keep the surfaces apart. It seems unlikely that the formation of adhesions could be prevented completely, for to do so the amount of air injected would have to be so great, in order to overcome the contraction of the scar tissue, that the pressure it would exert on the heart would be more than the latter could tolerate without failure. Possibly if a systematic attempt had been made to keep the pressure up in the case here reported, the adhesions would have been much less dense and the operation correspondingly easier and more successful.

CASE REPORT

A Portuguese boy of eighteen years came to the emergency ward of the Massachusetts General Hospital on August 26, 1930, complaining of attacks of vomiting for eight months and extreme shortness of breath for a few days. Family and past histories were irrelevant. There was no family history of tuberculosis.

The patient had always been very healthy and vigorous until shortly after a boxing bout eight months previously, when he developed mild epigastric and right upper quadrant, gnawing pain and tenderness associated with vomiting, which occurred a few minutes after meals. About the same time he developed a dull pain in the lower right chest which was most noticeable on leaning forward. He became rather short of breath and after two weeks or so of these symptoms he went to St. Luke's Hospital in New Bedford on December 28, 1929. A letter from this hospital states, "Physical examination of the heart at time of his admission showed the following: Right border 10 cm. to right of sternum by percussion, left border in axillary line. All this area is dull to percussion. Heart sounds are weak and quick. Rate 96. X-ray revealed (December 28, 1929) no evidence of fluid in chest. Heart shadow appears to be large, could be pericardial effusion. On January 1, 1930, heart shadow apparently smaller. Increased density in right chest from fourth rib in front to base. Diagnosis, pericarditis, acute, serofibrinous." No tap was done, nor was any specific treatment given other than general medical care. He improved and was discharged from St. Luke's Hospital on January 15, 1930.

Following discharge he continued to improve, had no more pain or vomiting and very gradually regained strength. The next two or three months he spent most of the time in bed, but as the spring advanced he gradually improved so that by June he was up all the time and did a little easy work such as raking lawns. He was, however, very short of breath, and whereas before his illness he had been able to swim several hundred yards without difficulty, he was now unable to swim more than ten strokes.

Two and one-half weeks before entry to this hospital the epigastric and right upper quadrant pain returned, accompanied by vomiting as before. A few days later there was swelling of the abdomen and some palpitation of the heart when lying down. For about two months he had had a cough with a small amount of sputum. He thought he had gained about twenty pounds during the illness.

The history was somewhat unreliable due to the limited intelligence of the patient.

Physical examination at entry showed a well-developed and nourished negro,

sitting up in bed, taking rapid, shallow breaths. The veins in the neck were somewhat distended. Cardiac dullness extended to the axilla on the left and nearly to the axilla on the right. The apex impulse was not felt. The supracardiac dullness was 6.5 cm. The heart sounds were faint but perceptible at the apex and fairly distinct at the base. There were no murmurs or rub. The pulse was 80 and definitely paradoxical. The blood pressure was 120/95 mm. The lungs were clear although the diaphragm was high on both sides. There was no Ewart's sign. The abdomen was tense and somewhat distended; there was a fluid wave and shifting dullness. The upper border of the liver was obscured by the cardiac dullness, but the edge was felt 4 cm. below the costal margin on the right and extended across the epigastrium. There was no edema of the genitals or extremities.

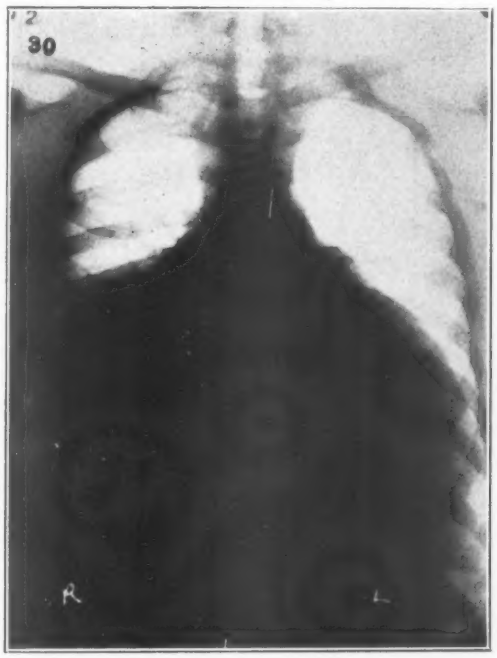

Fig. 1.—August 26, 1930. Shows the enormous size of the pericardial shadow on entry.

Laboratory findings on admission were a red blood cell count of 4,300,000, hemoglobin 85 per cent (Tahlquist), white blood cell count 9,550, and blood smear not remarkable. The urine was negative.

The day after entry x-ray picture of the chest showed an enormous heart shadow extending almost to the axillary line on both sides. The cardiac pulsation could not be made out by the fluoroscope. The right costophrenic angle was obliterated. The appearance suggested a pericardial effusion with a small amount of fluid in the right pleural cavity. A chest plate taken when the patient was on his back showed only a slight increase in the width of the supracardiac shadow.

The consulting cardiologist, Dr. Paul D. White, thought that pericardial effusion, probably due to tuberculosis, was the most likely diagnosis and advised tapping.

On August 28, 1930, taps were done in the fifth left interspace and the fourth right interspace; both yielded what was thought to be blood, but the fluid was not examined at the time, and this neglect caused us to abandon the diagnosis of

pericardial effusion. The same day he became much worse; he was very orthopneic, had more cough with some frothy white sputum (repeated examination of the sputum was always negative for tubercle bacilli); he was put on the danger list. He required considerable morphine to control the dyspnea. The abdominal discomfort and vomiting became much worse. Venesection of 200 c.c. on August 29 gave slight relief. Digitalization was started and was complete by September 1, with resulting decrease in the orthopnea.

For the next two and one-half weeks he ran an up-and-down course, fairly comfortable at times and at others extremely orthopneic with much pain, nausea and vomiting. Abdominal paracentesis on September 9 yielded only 5 c.c. of yellow fluid which clotted before it could be examined. Culture of this was negative,

Fig. 2.—October 5, 1930. This plate was taken with the patient lying on his right side immediately after the first air injection. The air rose to the highest part of the pericardial cavity and moved freely from one part to another as was shown by plates in different positions.

and a guinea pig injected with it showed no tuberculosis at autopsy six weeks later. He received salyrgan intravenously every three to four days which caused moderate diuresis. Prior to this treatment he had gained 6 pound since admission (154 to 160); after it he lost 14 pounds.

On September 17, 1930, another pericardial tap was done and 120 c.c. of bloody fluid removed which had red and white blood cell counts about one-third that of blood taken at approximately the same time. The following day 450 c.c. more were removed. After this he had no orthopnea and was subjectively much improved. On September 20 and 26, 270 and 600 c.c. of fluid respectively were removed from the pericardium causing little noticeable clinical change in spite of marked subjective relief. For a time after this he refused to stay in bed, but soon pain in the lower right axilla developed (without physical signs), and he returned to bed of his own accord.

After considerable discussion it was decided that air be injected into the pericardium the next time fluid was withdrawn. On October 5, 375 c.c. of fluid were removed by the sub-xyphoid route and 200 c.c. of oxygen injected. He stood the procedure well. As each 50 c.c. of fluid was withdrawn the blood pressure rose

about 5 to 10 points, and when a similar amount of oxygen was injected, it fell about the same amount. The pressure of the fluid in the pericardium was measured by a manometer connected to the injecting needle; this varied from 8 to 20 cm. of pericardial fluid, the high points occurring when the patient strained. Pulse and respiratory oscillations were visible, the respiratory oscillations being only 1 to 2 cm. and the pulse barely visible. (The manometer was connected with the needle by approximately two feet of tubing which probably had a definitely dampening effect on the oscillations.)

X-ray pictures taken after the tap showed gas in the pericardium, and plates taken with the patient in different positions showed that the cavity was apparently continuous and the pericardium not especially thick. No succussion splash or "bruit

Fig. 3.—October 10, 1930. Taken after the third injection of air, shows the pericardium distended with air, and the heart not definitely enlarged. There is fluid in the right pleural cavity.

de moulin" was audible after this tap, although resonance could be demonstrated by percussion.

Digitalis was omitted at this time because the patient refused to take it.

Since this first injection of gas resulted in no apparent ill effect, 740 c.c. more of fluid were removed and replaced by 590 c.c. of air on October 8, and 740 c.c. of fluid by 560 c.c. of air on October 10. During these taps venous pressure readings were taken which showed a definite fall when fluid was removed and a rise when air was injected. The venous pressure was always from 2 to 4 cm. of water greater than the pericardial pressure. Following the October 8th injection a succussion splash was heard, and the cardiac dullness was replaced by tympany. On October 11 he had marked respiratory distress, cough and abdominal pain which was not relieved by morphine, so 175 c.c. of air were removed. This came out under some pressure (not measured) and was followed by definite subjective relief. Several days later he developed pitting edema of the ankles which lasted for a short time, but he seemed to be much more comfortable and improved steadily. The liver edge,

which at first had moved lower until it reached the iliac crest, now began to recede and became much less tender. X-ray pictures taken two weeks after the last tap showed that approximately two-thirds of the air was still present. On October 29 a liver function test (tetra-bromsulphonephthalein) showed no retention of the dye, while on August 26 and September 15 there had been 15 per cent retention. On November 2 he developed a pericardial friction rub.

He continued to improve, but as the x-ray picture showed that considerable fluid was still present, another tap was done on November 12. Only 25 c.c. of fluid were obtained, and, although there was undoubtedly more fluid there, further attempts to withdraw it were not made because the patient became very uncomfortable and upset. Following this tap he had a chill and fever amounting to 103.5° F., but the next day he again felt better.

An x-ray picture of the chest on November 22 showed that the air had been entirely absorbed but that fluid was still present or had reaccumulated, so he was tapped again the next day, this time in the fifth intercostal space on the right, and 320 c.c. of fluid removed and 250 c.c. of air injected. This tap also was followed by a chill and fever.

From November 21 to December 6 he was given 0.2 gm. of sodium cacodylate three times daily in the hope that it might hasten the absorption of the fluid.

Following this last tap he continued to improve until he was discharged on February 7, 1931. Taps were attempted on December 20 and 24, 1930, both failing to yield fluid. His temperature, which had been swinging from 99° to 101° F. by mouth the first three months he was in the hospital, was rarely over 100° F. by rectum in December. From the last of December until discharge the temperature was taken by mouth and showed, until the middle of January, an afternoon rise to 99°, after which it was usually 98.6° or less.

During the patient's stay in the hospital nine sputum tests were negative for tubercle bacilli, the von Pirquet test was negative, and dilute intradermal tuberculin tests were negative. Three guinea pigs injected with pericardial fluid, one with abdominal fluid, and one with sputum, were negative for tuberculosis at autopsy.

The week before discharge a final x-ray picture showed that the heart was slightly enlarged and that there perhaps was a small amount of air along the left border of the heart; fluid was not demonstrable, although there was a rounded area of dullness along the right border of the heart. The pericardium appeared about one-quarter of an inch thick; it was quite thin in pictures taken earlier in the course of the disease.

The patient was readmitted to the hospital on March 23, 1931, six weeks after discharge. In the interval he had grown stronger, gained three pounds, and was a little less short of breath. He had had almost continuous, right upper quadrant pain and soreness with three attacks of rather acute right upper quadrant pain radiating to the left shoulder and left side of the neck.

Physical examination showed very little change from his condition at time of discharge, although he appeared definitely stronger. The heart sounds were rather muffled but of fair quality. Blood pressure was 110/75 mm. The veins of the neck were distended. The pulse was paradoxical. The lungs showed a small amount of fluid at the right base. The abdomen was somewhat distended; dullness in the flanks and a fluid wave were present. The liver edge was palpable 6 to 8 cm. below the costal margin and was rather tender. There was no edema of the genitals or extremities.

X-ray pictures showed that the cardiac shadow had decreased somewhat in size. Stereoscopic films showed that the rounded area of dullness at the right side of the heart, about 3 cm. in diameter, was a small interlobar collection of fluid, not connected with the pericardium.

During a two weeks' period of observation his temperature was normal, pulse about 80, urine negative except for an occasional, very slight trace of albumin, and white blood cell count slightly elevated, 10,000 to 12,000. In view of the persistent engorgement of the liver and ascites it seemed probable that he had an adherent pericardium which was interfering with the action of the heart, and since there seemed to be nothing to gain by further rest and general care, cardiolysis was decided upon. This was done on April 16, 1931, by Dr. Wyman Whittemore. Sections of the third to seventh costal cartilages on the left were removed. The pericardium, one-third to one-quarter of an inch thick, was adherent to the heart, although a definite line of cleavage was present. The pericardium was gently peeled off the heart and large flaps cut away, first on the left side, then on the right. Posteriorly, particularly near the apex, the pericardium was more adherent and could not be removed. When the anterior portion of the thickened pericardium was

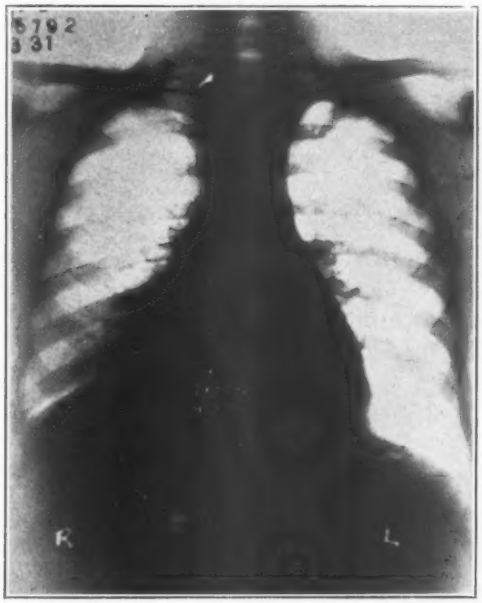

Fig. 4.—April 3, 1931. Taken two weeks before cardiolysis. The dullness at the right border of the heart was shown stereoscopically to be probably a small interlobar collection of fluid.

removed, the heart bulged into the opening thus formed; it seemed evident that its activity was much less hampered.

Pathological examination of the pieces of pericardium removed showed in one area a focal collection of epitheloid cells and lymphocytes, the appearance of which was sufficiently typical to justify a diagnosis of tuberculosis in the healing stage.

After the operation, which caused relatively slight shock, first serum drained from the incision, later pus from two sinuses in the scar. The patient ran an afternoon temperature of 100° to 101° by rectum until the middle of June. His pulse, still paradoxical, at first was 100 to 120, and later fell to 90 to 100. The white blood cell count varied from 11,000 to 15,000. The blood pressure remained low, 85-90/70. Digitalization had no definite effect on either the pulse or the urinary output, although with salyrgan a fair diuresis was obtained several times without causing any perceptible difference in the amount of fluid in the abdomen and chest.

His general condition and strength, however, gradually improved so that on June 26 he was discharged to Lakeville Sanatarium for general care. At the time of his discharge his condition was not so good as before operation; apparently the quiescent tuberculous process had become active again. It seemed likely that adhesions in the region of the right auricle and inferior vena cava were responsible for the continued ascites and other signs of venous obstruction, but further operative procedures in the presence of probably active tuberculosis seemed contraindicated.

A report from the Lakeville Sanatarium on October 30, 1931, states that the patient's general condition is good. At time of admission to that institution he was dyspneic and had considerable precordial pain. He was kept in bed and given one and one-half grains of digitalis daily with resulting disappearance of the dyspnea and precordial pain. He still had right upper quadrant pain.

Physical examination at the time of this report revealed regular heart sounds of good quality, rate 70 to 80. The liver edge was still palpable four to five finger-breadths below the costal margin, but there was no abdominal tenderness. There was still slight drainage from the sinus in the operative scar. His temperature had remained normal and the white blood count had been 8,000.

X-ray picture of the chest showed no essential change from that taken just prior to operation.

He had gradually increased his activities and was up and about for short periods daily.

SUMMARY

A brief review of the literature on therapeutic pneumopericardium is presented. A case of tuberculous pericarditis with effusion treated first by pneumopericardium then by cardiolysis is reported. From the present case and the few cases now in the literature it is impossible accurately to estimate the therapeutic value of artificial pneumopericardium. It did not cure this patient nor even prevent the necessity for operation later. This may have been because the treatment was not continued for a sufficiently long period. If one wishes to prevent the formation of pericardial adhesions in pericarditis, it would seem, on theoretical grounds, preferable to keep the parietal and visceral pericardium apart by means of elastic gas rather than by inelastic fluid. The method of artificial pneumopericardium therefore would seem to deserve more extensive trial than it has yet been accorded.

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ANEURYSM OF THE AORTA PRODUCING PULMONARY STENOSIS AND BUNDLE-BRANCH BLOCK*

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IT IS uncommon for an aneurysm of the aorta to encroach upon the pulmonary artery, conus arteriosus, or right auricle, or to invade the interventricular septum. In some instances such aneurysms are of congenital origin being due to defective development of the bulbar septum and are, in consequence, often accompanied by interventricular septal defects. These aneurysms are usually small. They arise from a sinus of Valsalva and project into the conus of the right ventricle where, as a rule, they finally rupture. Abbott,[1] in reporting a case of this nature, reviewed the literature and discussed the developmental defects which cause the condition.

Acquired aneurysms of this group are relatively more frequent and usually follow luetic aortic disease. Their common site of origin is higher than that of the congenital type and they more commonly project into the pulmonary artery than into the right ventricle. Scott,[2] in 1924, described two cases in which rupture into the pulmonary artery had occurred and reviewed the literature of the acquired type up to that date. Since then a few additional cases have been reported. Gunn[3] described an aortic aneurysm which appeared to have produced compression of the pulmonary artery into which it finally ruptured. Rothschild, Sacks, and Libman,[4] in a discussion of the disturbances of cardiac mechanism in subacute bacterial endocarditis and rheumatic fever, mention briefly one case in which a mycotic aneurysm of a sinus of Valsalva projected into the interventricular septum and caused partial bundle-branch block. They state that serial sections demonstrated that the aneurysm had partially intercepted the path of the left branch of the bundle of His. Buffalini[5] reported a case in which two aneurysms arose from the first part of the aorta and encircled the pulmonary artery, thus causing stenosis. This case was presented in greater detail by Costa[6] who also described another in which an aneurysm had perforated into the pulmonary artery. Laederich and Poumeau-Delille[7] reported an aneurysm which projected into the right auricle where it finally ruptured. The case of Stejfa[8] in which an aneurysm the size of a walnut caused compression of the pulmonary artery and also, he believed, block of the "left branch of Tawara's node" will be discussed in greater detail later. Schwab and Sanders[9] reported a case of acquired aneurysm which had ruptured into the right ventricle and they were able

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to find only two previous cases of this nature in which such an occurrence had taken place. The following case is presented because of the unusual growth direction taken by the aneurysm and because of interesting pathological changes which it appeared to have caused.

CASE REPORT

History.—R. L., an intelligent colored man, aged thirty-six years, a motor mechanic by occupation, came to the Cardiac Clinic of the Long Island College Hospital on June 5, 1929. He complained of dyspnea, palpitation, weakness, abdominal swelling and constipation. Five years previously, he commenced to suffer from dyspnea on exertion, marked palpitation and weakness, and he received medical treatment which relieved him to some degree. During the last six months all of these symptoms had become progressively worse and the patient noticed that his abdomen had been considerably distended. Dizziness had been gradually increasing during the past five months and he had fainted on one occasion. On admission to the Clinic he could walk only a short distance without marked discomfort. Both dyspnea and palpitation were extremely marked on any exertion but precordial pain was absent.

Previous Illnesses.—At the age of sixteen the patient suffered from gonorrhea and syphilis with secondary manifestations. He received a series of mercurial injections in a hospital in Philadelphia but on leaving the hospital he discontinued treatment.

Family History.—Nothing of note except that his wife had never been pregnant.

Physical Examination.—The patient was a well built colored man weighing 195 pounds. Even slight exertion caused considerable dyspnea. Slight cyanosis of the lips was present but there was no edema. The pupils were equal and reacted to light. Marked pulsation of the vessels of the neck was seen, the arterial pulsation being much more marked than the venous. The radial pulse rate was 72 beats per minute with some irregularity due to extrasystoles. Both pulses were equal and these was evidence of sclerosis of the peripheral vessels. The blood pressure was 130/65 mm. A suggestion of a capillary pulse was present but no peripheral signs of aneurysm were elicited.

Heart.—There was a heaving diffuse pulsation all over the precordium and the apex beat was felt in the 6th interspace 15 cm. from the midsternal line. No thrill was present over any area. The heart was markedly enlarged both to the left and to the right and on the left side there was a slight prominence in the region of the pulmonary conus and left auricle. The aortic arch did not appear widened. On auscultation there was noted a very loud, rasping systolic murmur, which was maximal in the second left interspace but could be heard over the whole precordium and also in the vessels in the neck. A diastolic murmur, of which the point of greatest intensity was in the second and third interspaces, was heard to the left of the sternum and was transmitted faintly over the rest of the precordial area. The second sound was almost inaudible over the entire precordium. An occasional râle was heard at the bases of the lungs. The liver was slightly enlarged. The nervous system showed no abnormality.

Electrocardiogram.—The auriculo-ventricular conduction time was slightly prolonged (0.22-0.24 sec.). A marked degree of partial right bundle-branch block (new terminology) was shown as the QRS complex was 0.15 sec., the main initial deflection was negative in Lead I and positive in Lead III with T opposite to the main deflection in each lead (Fig. 1). An irregularity, due to extrasystoles which appeared to arise in the upper septal region of the ventricle, was also found.

X-Ray and Fluoroscopy.—The heart was tremendously enlarged both to the right and to the left. (Cardiac Index 0.8.) The right side was of a globular shape and the left side was unusually straight with a slight prominence in the region of the pulmonary conus and left auricular appendix. The aortic arch was not widened and no evidence of aneurysm was seen. (Fig. 2.) On fluoroscopy no aneurysm was ap-

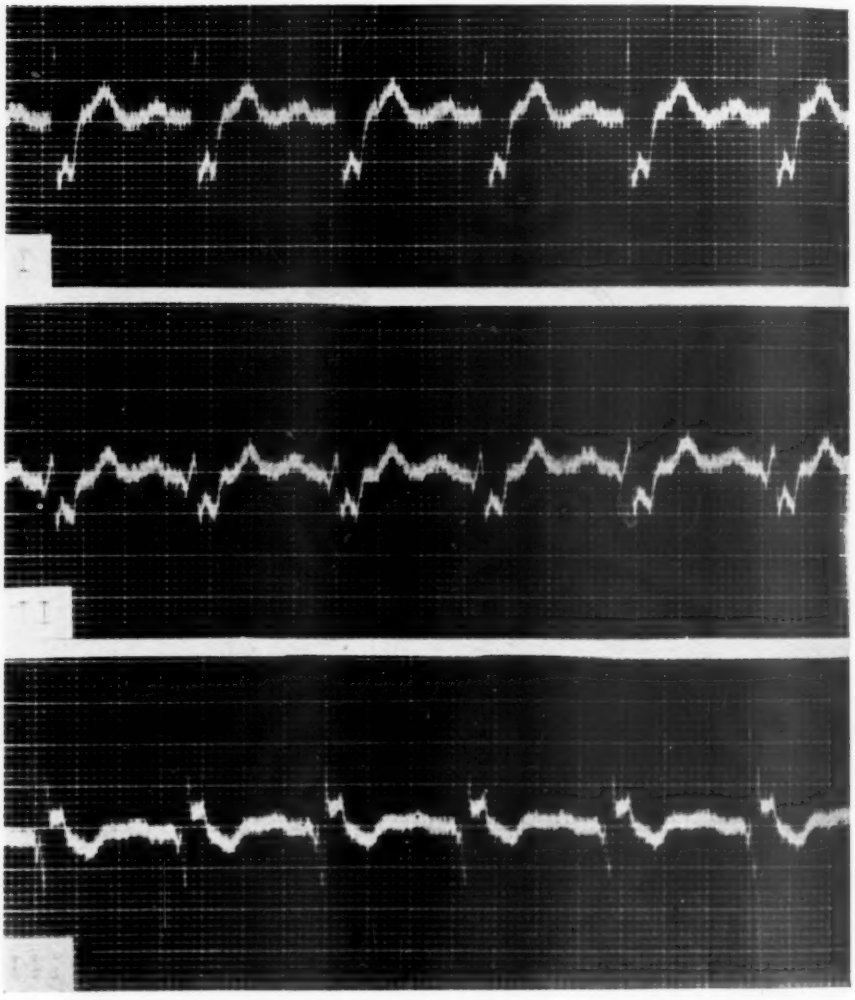

Fig. 1.—Electrocardiogram taken on July 2, 1929, indicating a high grade of bundle-branch block.

parent and the posterior mediastinal space did not appear to be encroached upon more than is usual with hearts of this size.

Laboratory Examinations.—The Wassermann test, although frequently repeated, was positive on only one occasion when it was +2. Other laboratory findings were normal.

The opinion was expressed that the patient was suffering from tertiary syphilis and aortic insufficiency with gummatous lesions producing the bundle-branch block.

As this did not appear to cover all of the findings, it was considered possible that congenital pulmonary stenosis was also present.

Treatment.—Complete rest in bed at home was ordered and mercury and potassium iodide were given by mouth.

Progress Notes.—As the patient did not show any improvement, he was admitted to the Long Island College Hospital on June 13, 1929. At this time the only change in the condition was that pulsus bigeminus was present. The site of origin of the extrasystoles as shown by the electrocardiogram had changed from the septal region to the wall of the right ventricle. The same treatment was continued and, in addition,

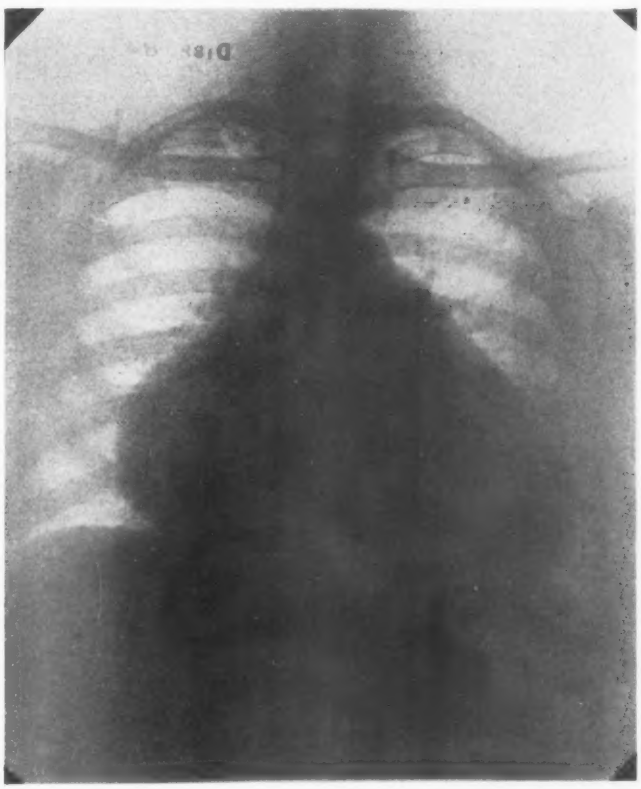

Fig. 2.—X-ray of heart (6 foot plate) taken on June 3, 1929, showing marked enlargement of both left and right ventricles.

the patient was fully digitalized. The extrasystoles soon disappeared and the patient slowly improved. The patient was discharged from the hospital on July 20, at which time he was able to take mild exercise without marked discomfort. He visited the Clinic at intervals during the next nine months but his condition became gradually worse until he finally became bedridden. On March 22, 1930 he was readmitted to the hospital. No marked dyspnea was noted while at rest and the heart showed no significant change since the previous admission. Fluid was present in the right pleural cavity and the liver was enlarged 12 cm. below the costal margin but there was no edema of the extremities. The x-ray and electrocardiographic findings at this time were the same as before. As the patient had been on a maintenance dose of digitalis, this was continued and potassium iodide was given in large

doses. At first there was some improvement in the condition and the fluid in the pleural cavity disappeared. Soon, however, he began to grow worse. The liver became progressively larger while edema of the extremities and ascites appeared and increased despite the administration of various diuretics. He died on May 6, 1930.

Autopsy No. 1340. May 6, 1930.—Marked edema of the extremities and of the tissues of the abdominal and thoracic walls was found at autopsy. A frothy, blood-tinged fluid exuded from the mouth and nose. There were otherwise no external abnormalities of note.

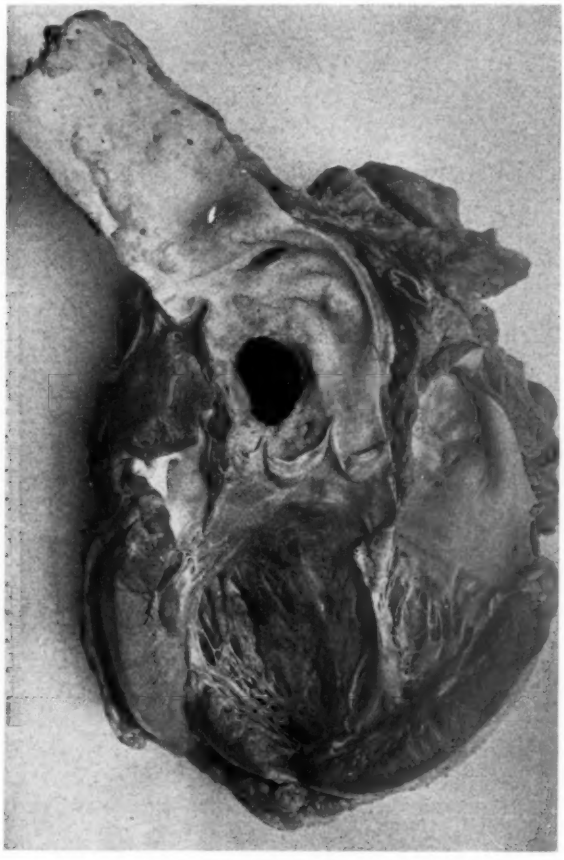

Fig. 3.—View of the left ventricle and aorta showing the orifice of the aneurysm. The luetic puckering of the aorta and the blotchy discoloration of the septum can be seen.

The pericardial sac was greatly enlarged, partly through the presence of about half a liter of clear, straw-colored transudate and partly because of great enlargement of the heart. The distended sac measured 20 cm. in its greatest transverse diameter. Both the parietal and visceral layers of the pericardium were markedly thickened and numerous recent adhesions were found at various points.

The heart was enormously enlarged, its weight being 975 grams. This enlargement was due to hypertrophy and dilatation of both ventricles and to a particularly marked dilatation of the right auricle. A firm rounded mass presenting on the anterior aspect of the heart between the right auricle and the conus arteriosus also contributed to the general enlargement.

On opening the chambers of the heart and the great vessels a saccular aneurysm of the ascending aorta was found. It was roughly spherical in shape, measuring 9 cm. in average diameter and was about half filled with lamellated thrombus. The orifice of the aneurysm was circular, 3 cm. in diameter, and the edges were rounded and smooth. Its lowest point was 1 cm. above the commissures of the right and left cusps of the aortic valve (Fig. 3). In its growth the aneurysm had pushed forward compressing the base of the pulmonary artery and the conus arteriosus. It had also pushed downward into the tissue of the interventricular septum and outward into the base of the right auricle. Superiorly it presented as a firm rounded mass between the pulmonary artery and the right auricular appendix.

The aorta measured 8 cm. in circumference at its base, which was the widest part. The ascending portion showed the typical scarring and intimal hyperplasia of luetic mesaortitis. The sinuses of Valsalva and the coronary orifices were not particularly involved in the luetic process. The aortic valve cusps were noticeably shortened in their vertical dimensions, moderately thickened, and the edges somewhat rolled. The

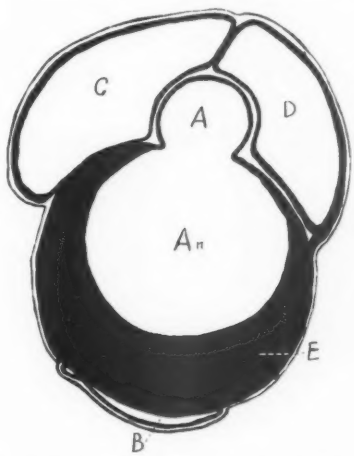

Fig. 4.—Diagrammatic representation of a cross section (roughly to scale) immediately above the aortic valve, showing compression of the pulmonary artery by the aneurysm. *A*, aorta; *An*, aneurysm; *B*, pulmonary artery; *C*, right auricle; *D*, left auricle; *E*, lamellated thrombus in the aneurysmal sac.

right and left cusps showed a 2 mm. separation at their commissures. The valve was incompetent to some extent.

The base of the pulmonary artery and the conus arteriosus had been so compressed by the aneurysm that a transverse section in the region of the pulmonary valve showed a crescent-shaped lumen with the opposing walls partially in contact with each other. At one point, immediately above the valve, there were organized adhesions between the opposing surfaces (Fig. 4). The right and left cusps of the pulmonary valve were tightly stretched over the bulging aneurysmal mass and appeared incapable of functioning. The portion of the aneurysm which presented in the conus consisted of a very hard rounded shell of connective tissue over which a glistening layer of endocardium was stretched. No remains of heart muscle were grossly visible in this area. Immediately below the left cusp of the valve the aneurysm showed a small, softened, necrotic patch, of a dirty gray-brown color contrasting sharply with the pale surrounding connective tissue. Perforation into the conus at this point seemed imminent (Fig. 5).

Both right and left ventricles were enormously hypertrophied and dilated. The right auricle was also markedly dilated while the left auricle was approximately normal in size. The tricuspid valve ring measured 13.5 cm. in circumference.

As noted above, the aneurysm had pressed down into the base of the interventricular septum near its anterior limit and in this situation a zone of "pressure atrophy" with fibrosis adjoined the wall of the aneurysm. Irregular grayish patches of fibrosis, reddish streaks of granulation tissue, and yellow-brown areas of recent necrosis were scattered throughout the remainder of the septum.

The coronary vessels were explored as far down as could be reached with fine scissors and probes but no thrombosed vessels were found. The anterior descending branch of the left coronary artery was markedly constricted as it curved around the

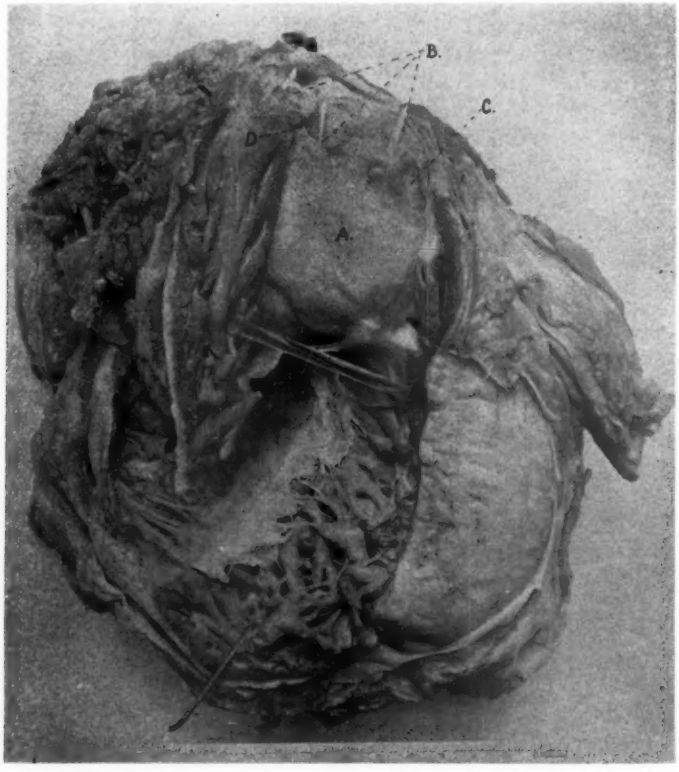

Fig. 5.—View of the right heart showing *A*, the aneurysm bulging into the conus; *B*, the distorted pulmonary valve cusps; *C*, the area of necrosis in the aneurysm; *D*, the fusion of opposing surfaces of the pulmonary artery.

aneurysm, while beyond this area it was of normal calibre. The first portion of the right coronary was similarly constricted.

The pathological changes in the rest of the body may be summarized as those of chronic passive congestion and anasarca. Death appeared to have been due to myocardial failure.

An attempt was made, through a study of serial sections of the septum, to identify the bundle of His and its main branches in order to determine the location of the block which had been electrocardiographically established. Sections were cut transversely through the entire upper portion of the septum. The wall of the aneurysm consisted of dense hyalinized connective tissue containing deposits of calcium salts. Merging with the aneurysmal wall was a zone of fibrosis in which but few remains

of heart muscle were found, while the remainder of the septum showed a considerable amount of scar tissue, granulation tissue, and heart muscle in various stages of degeneration and necrosis. Occasional small granulomatous lesions, interpreted as being miliary gummata, and extensive small round-cell infiltration were also found. Because of the extensive necrosis and fibrosis the conduction pathways could not be identified with certainty.

The microscopic sections of the ascending aorta showed typical mesaortitis luetica while slight scarring, marked edema, cloudy swelling, and hydropic degeneration were seen in those from the ventricular walls.

DISCUSSION

In the reported cases of aortic aneurysm projecting into the pulmonary artery or right ventricle, both congenital and acquired, the diagnosis rarely has been made before rupture. Indeed, except in the congenital cases with an associated septal defect, it was unusual for symptoms to be present before perforation had taken place. After this event there appeared pathognomonic symptoms, which have been described fully both by Abbott[1] and by Scott.[2] Only two cases have been found which closely resembled that described above, one by Rothschild and his coworkers[4] and another by Stejfa.[8] The former was only incidentally reported with few details while the latter was fully discussed. Stejfa made a diagnosis of aortic aneurysm compressing the pulmonary artery on the basis of a rough systolic murmur heard in the second and third left interspaces with enlargement of the heart to right and left. In addition, there were signs of aortic insufficiency while a tracheal tug and paralysis of the left recurrent laryngeal nerve were present. X-ray examination showed only a dilated aorta and cardiac enlargement. He believed that these signs could be explained only by the presence of an aneurysm compressing the pulmonary artery. From his electrocardiographic studies he concluded that there was also a block of the "left branch of Tawara's node." One electrocardiogram was illustrated with the interpretation of which we disagree. It does not appear to us to fulfill the criteria necessary for a diagnosis of bundle-branch block but rather indicates marked right axis deviation which could be explained easily by pulmonary stenosis with right ventricular hypertrophy. At autopsy an aneurysm the size of a walnut was found. It projected into the pulmonary artery causing high degree stenosis but in view of its size it appeared to be situated too high up to have damaged the septum. The aorta showed signs of lues and was dilated while the aortic valve was incompetent. The description of the studies of the bundle appear too vague to permit definite conclusions.

The case which we are reporting was studied thoroughly both clinically and by x-ray but aortic aneurysm was not suspected, as there was no evidence on which such a diagnosis could be supported. As there was no rupture the characteristic physical signs of communication between the aorta and the pulmonary artery or right ventricle were lacking. The

rough systolic murmur heard best in the second left interspace and the marked globular shape of the right heart on x-ray lead us to suspect that a lesion of the aortic valve did not cover the complete diagnosis and congenital pulmonary stenosis was considered an additional possibility. At autopsy, stenosis of the pulmonary artery and conus, due to compression by the aneurysm, was found and incompetence of the pulmonary valve had been produced. It seems probable, in view of the relatively slight change in the aortic valve and the paucity of peripheral signs of aortic insufficiency, that the diastolic murmur was really due to pulmonary incompetence.

The electrocardiographic findings were of considerable interest in that the tracings showed partial bundle-branch block in which the chief initial deflection was downward in Lead I and upward in Lead III. For many years, based on the experimental work of Lewis and his associates,[10, 11] this was considered to represent a lesion of the left division of the bundle of His. Fahr,[12] on theoretical grounds, called in question this interpretation and believed that the curves ascribed to right bundle-branch block by Lewis were really curves of left bundle-branch block and vice versa. Barker, Macleod, and Alexander,[13] as a result of their study of extrasystoles in the exposed human heart, brought forward evidence which strongly supported the latter view and, more recently, other workers, both on theoretical[14, 15] and experimental[16, 17] grounds, have come to the same conclusion. The present case cannot furnish conclusive evidence on this point as extensive necrosis of the septum was found and the divisions of the bundle could not be traced by serial sections. However, the facts that the aneurysm projected into the right ventricle and that the damage to the septum was greater on the right side make it probable that the right, rather than the left division of the bundle was damaged, thus tending to support the newer conception. Also, assuming that the bundle of His pursued its normal path, the point of penetration of the septum and subsequent course of the left division would be below and posterior to the aneurysm, while the region traversed by the right division in its course to the papillary muscle would be invaded by the lower pole of the aneurysmal sac.

Several causative factors appeared to be concerned in the extensive damage to the septum. The aneurysm, pushing downward into the base of the septum, caused considerable atrophy and fibrosis in the subjacent tissue. There were also a number of small gummata and extensive areas of recent necrosis. Although the main branches of the coronary vessels showed no evidence of thrombosis, they were markedly compressed where they skirted the aneurysm. Some of the degenerative myocardial changes might be ascribed to this compression with resulting restriction of the blood supply. This must at least have played an important part in the cardiac decompensation.

SUMMARY

A case is reported of a patient suffering from luetic aortitis in whom an aneurysm 9 cm. in diameter, arising in the first portion of the ascending aorta, had produced none of the usual clinical evidences of aortic aneurysm. A high grade bundle-branch block was known to have been present for a period of eleven months prior to death from myocardial failure. The aneurysm, which had not ruptured, caused a marked stenosis of the pulmonary artery and insufficiency of the pulmonary valve. It projected into the right ventricle and produced considerable damage to the interventricular septum. This appeared to be the most important factor in the causation of the bundle-branch block. Slight aortic insufficiency was also present.

We wish to thank Dr. Alfred E. Cohn for his assistance in the study of the bundle of His.

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BRAIN ABSCESS (PARADOXICAL) IN CONGENITAL HEART DISEASE*

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ALTHOUGH the older English students recognized cerebral abscess as part of the classic picture of congenital heart disease,[1] nevertheless relatively few cases, confirmed by postmortem examination, have been reported since Ballet[2] in 1880 first definitely assumed a causal connection between this cerebral lesion and congenital cardiac conditions. Abbott, Lewis and Beattie[1] in 1923, after a careful search of the literature collected 14 cases of paradoxical cerebral embolism, 6 of which proved to be cerebral abscesses. We have been able to find only 4 additional cases reported up to the present time. We wish therefore to record this case which we had the opportunity of observing.

CASE REPORT

T. S. (Case No. 124623), female, aged sixteen and one-half years, Hebrew, schoolgirl, was seen in consultation at her home on November 24, 1929 by one of us (M. A. R.) who, because of the story of a sudden onset of cerebral symptoms, fever, and the presence of a congenital cardiac lesion, made the diagnosis of a paradoxical brain abscess, and sent her into the wards of the Jewish Hospital of Brooklyn, where the following history was recorded. Her chief complaints at the time of admission were headache and vomiting of one week's duration. Her family history was essentially negative. Her past history revealed measles during infancy, tonsillectomy at the age of seven, and the fact that it was known that she had had a cardiac lesion since infancy. There was no history of cyanosis, episodes of cardiac failure, or embolic phenomena. The patient was apparently well until one week before admission, when, following a large and indigestible meal she became ill with abdominal discomfort and malaise. The next morning she awoke with a severe generalized headache which became associated on the following day with vomiting, not projectile in character, and often induced by the patient. The headache grew progressively worse, and at the time of admission to the hospital was localized occipitally and described as "terrific and unbearable." There was no history of fever, sweats, convulsions, twitchings, or paralyses.

Physical examination revealed a female of about sixteen years of age, well nourished and developed, acutely ill, very restless, thrashing about in bed, face flushed, lips cyanotic. The scalp was normal. Tenderness was elicited over the occipital region. The eye examination revealed the nasal half of the right disc to be blurred and indistinct, the temporal half showing beginning blurring, and a swelling of the disc of one diopter; the left disc showed a swelling of 3 diopters; the veins were distended and tortuous; there were no hemorrhages or exudate. The ears and nose were negative. The tongue protruded in the midline. The teeth were in good condition. There was no rigidity of the neck. Percussion of the heart showed enlargement to the left. There was a rough systolic murmur most marked at the apex, but heard over the entire precordium and at the base. The sounds were of good quality and regular in rhythm. The abdomen revealed no tenderness or masses, the liver and spleen were not felt. The extremities were negative, except for cyanosis

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of the finger tips and toes. The deep reflexes of the upper extremities were sluggish. The abdominal reflexes were sluggish; the knee jerks very sluggish; the ankle jerks active. The Brudzinski and Kernig signs were negative. The Babinski and Hoffmann signs were bilaterally positive.

On admission the temperature was 99.4° F., pulse 86, respirations 20. The white blood cell count showed 17,100 leucocytes, 88 per cent polymorphonuclears, 4 per cent mononuclears and 8 per cent large lymphocytes. The red cell count was 6,120,000 with 100 per cent hemoglobin. The blood pressure was 100 mm. systolic and 70 diastolic. Spinal puncture revealed fluid under markedly increased pressure, with 20 lymphocytes per c. mm., and chemical examination showed sugar 93 mg. per 100 c.c., albumin 4 plus, globulin 3 plus; no tubercle bacilli were found. The colloidal gold curve was 0000133320. The blood Wassermann and Kahn reactions were negative.

The patient was admitted to the hospital at 9:30 P.M. She received magnesium sulphate solution intramuscularly and 50 per cent glucose intravenously. She appeared quieter and more comfortable, but died suddenly at 7 A.M. the next day.

Postmortem Examination.—General: Body was that of a well-developed and well-nourished female about sixteen years of age.

Cavities showed no abnormalities.

Lungs were voluminous. On section there oozed from the cut surface a large amount of bluish red frothy material. Throughout the parenchyma were scattered many hemorrhagic areas about 4 mm. in diameter. The bronchi and vessels appeared normal. Microscopically the lung tissue was markedly congested, and edematous. The alveoli contained numerous red blood cells.

Heart weighed 340 grams, was pointed and contracted. The surface was dull and the pericardium was opaque in spots. The right auricle was normal. The foramen ovale contained two funnel-shaped openings. The right ventricle was enormously thickened, the wall measuring about 1 cm. The accessory musculature was hypertrophied. The tricuspid valve was normal. The left auricle was normal in size. The wall of the left ventricle was about 4 mm. thick and appeared normal. The mitral valve was normal. The septum membranacea and the upper portion of the intraventricular septum were missing. The upper border of the septum was represented by a semilunar rim with a concavity facing upward. The aorta originated from the right, while the pulmonary artery arose from the left side of the common ventricular orifice. The pulmonary ostium was decidedly smaller than normal. The ductus arteriosus was not patent. The aortic and pulmonary valves were normal.

Esophagus and stomach showed some submucous hemorrhages.

Intestines appeared normal. The papilla of Vater was patent.

Pancreas weighed 79 grams and appeared normal, macroscopically and microscopically.

Spleen weighed 118 grams. Capsule was smooth, color was deep purple, consistency normal. On section parenchyma was deep blue in color, the follicles were large and prominent. On the anterior perirenal tissue there was found an accessory spleen, 1.5 cm. in diameter, resembling on section the splenic tissue. Microscopically the spleen and accessory spleen showed congestion and hemorrhage in the pulp with endothelial hyperplasia.

Adrenals weighed 13 grams each, and showed no abnormalities. Microscopic examination revealed an adenoma.

Kidneys weighed 107 and 122 grams. They were normal in size and shape. Capsule stripped easily, leaving a smooth, clean surface. On section they appeared normal except for a deep blue red appearance. Microscopic examination showed congestion and extensive hemorrhages in the collecting tubules.

Ureters and bladder were normal.

Uterus was soft, slightly enlarged. Mucosa was thick and red (menstrual). Microscopic examination of ovaries and uterus showed them to be normal.

Bone marrow was normal.

Liver weighed 1300 grams; on microscopic examination it appeared normal.

The ears and sinuses were normal. The calvarium contained a number of thin areas of varying size through which light passed (osteogenesis imperfecta).

Brain weighed 1275 grams. Dura was normal. The entire brain was markedly edematous, the right side more than the left. The convolutions were very prominent. The optic nerves appeared flattened. In the cerebellum there was an abscess cavity, of the size of a walnut, containing foul smelling pus. On culture this pus showed Bacillus coli. The membranes were normal. The pituitary body was microscopically normal.

The findings in the heart in this case on postmortem examination present the typical combination of congenital cardiac anomalies known as the "Tetralogy of Fallot,"[3] namely pulmonary stenosis, ventricular septal defect, dextroposition of the aorta and hypertrophy of the right ventricle. Although, as was brought out by Baumgartner and Abbott,[4] this is the most common of all cardiac abnormalities in patients with congenital cyanosis reaching adult life, this patient gave no previous history of cyanosis or signs of cardiac embarrassment.

The source of the infected embolus is not clear in this case. The onset of the illness with gastro-intestinal symptoms and the finding of foul smelling pus in the cerebellar abscess cavity, which showed the colon bacillus on culture, suggest the possibility of the gastro-intestinal tract as the point of origin.

DISCUSSION

The history of the study of the mechanism of the paradoxical brain abscess applies of course also to the paradoxical embolus without the infective factor. Abbott, Lewis, and Beattie[1] reviewed the work of Cohnheim (1877) who was the first definitely to trace the path of an embolus through an opening in the cardiac septum, and whose findings were later confirmed by Virchow (1880), Zahn[5] (1881), Rostan[6] (1884), and Hauser[7] (1888). Ohm[8] in 1907 reported a case of hemorrhoidal vein thrombosis which gave rise to repeated cerebral emboli in a case of patent foramen ovale. He discussed in detail the mechanism involved.

The brain abscess is the result of a primarily infected embolus or, less frequently, of secondary infection after embolization has occurred.

While we find the occurrence of the paradoxical cerebral abscess most often in the congenital cardiac patient presenting the combination known as the "Tetralogy of Fallot," the occurrence is possible in any case where a communication exists between the right and left sides of the heart. Baumgartner and Abbott[4] report a case occurring with a much less common combination of congenital cardiac anomalies, known as the "Eisenmenger complex," described by Abbott,[9] which differs from the "Tetralogy of Fallot" in that there exists a dilatation instead of a stenosis of the pulmonary artery. Ballet's case[2] presented only the interventricular septal defect without the other anomalies of the

"tetralogy." Ballet quoted the case of Lallemand in which there was a patent foramen ovale, pulmonary stenosis, and no ventricular septal defect.

The 10 cases which we have reviewed are classified according to the cardiac anomalies which existed and summarized as follows:

I. DEFECT OF THE INTERVENTRICULAR SEPTUM

A. Resembling the "Tetralogy of Fallot."

1. By J. R. Farre[10] (1814). Male, aged nine and one-half years. Cyanosis observed definitely from the age of two and one-half. Four days before death, left hemiplegia, with severe pains in head, fever, and rapid pulse rate. Autopsy findings: Ventricular septum defect, pulmonary stenosis, deviation of aorta to the right. Abscess in right hemisphere of brain, containing one-half ounce of thick pus.

2. By Charles Bertody[11] (1845). Female, aged twenty-one years, complained of slight cyanosis and dyspnea on exertion through life. Always suffered from headaches, which a few days before death became markedly increased and were associated with fever and later with delirium and coma. Autopsy findings: Ventricular septum defect at orifice of aorta. Pulmonary artery contracted and aorta arising from both ventricles. Abscess in left posterior lobe of brain, size of a pigeon's egg; left lateral ventricle filled with pus.

3. By W. H. Stone[12] (1881). Female, nineteen years old. Cyanosis and dyspnea from birth. Sudden onset of severe headache shortly before death. Autopsy findings: Conus stenosis of right ventricle; acute endocarditis of lower conus orifice. Large ventricular septum defect at base below aortic orifice. Right ventricular wall hypertrophy. Purulent meningitis. Cerebral abscess in right occipital lobe, containing fetid pus, which had burst into the horn of the right lateral ventricle.

4. By Th. Deneke[13] (1906). Male, aged eighteen years. Cyanosis and clubbing of fingers since childhood. Ten days before death sudden onset of severe headaches and left-sided hemiplegia. Autopsy findings: Large ventricular septum defect. Transposition of aorta and narrowed pulmonary artery. Right ventricular hypertrophy. Narrow ductus Botalli. Streptococcic abscess of right cerebral hemisphere, size of walnut.

5. By Abbott, Lewis and Beattie[1] (1923). Male, aged eleven years. Cyanosis and dyspnea from birth. Appendectomy for acute appendicitis eight days before death. Six days after appendectomy sudden onset of severe headache and temperature of 101° F. On following day right hemiplegia. Autopsy findings: Pulmonary atresia, ventricular septum defect at base. Aorta arising from conus of right ventricle. Hypertrophied right ventricle. Cerebral abscess of left frontal lobe. Purulent meningitis.

6. By William Raab[14] (1923). Male, aged fifteen years. Cyanosis and dyspnea since attack of scarlet fever at age of six. Sudden onset of violent frontal headaches and fever to 104° F. three weeks before death. One week before death, left hemiplegia. Autopsy findings: Hypoplasia of pulmonary artery with stenosis of orifice. Ventricular septum defect, patent foramen ovale. Right ventricular hypertrophy. Aorta arising from both ventricles. Recrudescing verrucous endocarditis of all valves of all orifices. Abscess, size of hen's egg, in right cerebral hemisphere under central fissure, and perforation of abscess into right lateral ventricle. Purulent meningitis.

7. By Frances Bach[15] (1928). Male, aged thirty years. Dyspnea and cyanosis since birth. For ten years fainting spells and severe frontal headaches on exertion. Fourteen days before death 6 carious teeth were removed. Five days later onset of severe frontal and temporal headaches. Temperature to 103° F. Headaches progressive. Coma. Autopsy findings: Tooth sockets healthy. Heart: Aorta arising from both ventricles; narrowed pulmonary artery; hypertrophied right ventricle; large

ventricular septum defect; open foramen ovale. Brain: Large abscess in right posterior temporal region; purulent meningitis.

B. Presenting the "Eisenmenger Complex."

8. By Baumgartner and Abbott[4] (1929). Male, aged twenty years. Moderate dyspnea and cyanosis on exertion all his life. About two weeks before death onset of headache. Later drowsiness, left hemiplegia, difficulty in speech, progressive headache, coma. Autopsy findings: Marked dilatation of pulmonary artery, aorta arose from both ventricles, ventricular septum defect, right ventricular hypertrophy. Large abscess in right frontoparietal region involving motor area and internal capsule. Culture of pus showed Streptococcus hemolyticus.

C. Ventricular Septum Defect With Anomalies Other Than the Above.

9. By Ballet[2] (1880). Male, aged fifteen years. No history of cyanosis. Left hemiplegia ten days before death. Autopsy findings: Ventricular septum defect. Malposed septum. Aplastic right ventricle. Both auricles opened into left ventricle which was markedly hypertrophied. Abscess, size of small orange, in frontal lobe, and purulent infiltration of surrounding brain tissue.

II. PATENT FORAMEN OVALE WITH PULMONARY STENOSIS AND CLOSED INTERVENTRICULAR SEPTUM

10. By Lallemand[16] (1821) (quoted by Ballet). Female, aged fifty-seven years. Violet-red color of face since infancy. Severe dyspnea on exertion. Left hemiplegia, twelve days before death. Autopsy findings: Patent foramen ovale, pulmonary artery stenosis, right auricular and right ventricular hypertrophy. Cerebral abscess, size of hen's egg, in right hemisphere containing 3 ounces of yellowish-green pus.

That the condition is more common than the paucity of the subject in the literature would lead one to believe is indicated by an analysis of 711 consecutive autopsies by Rostan.[6] He found a patent foramen ovale 139 times, or in 20 per cent of the cases; and in 7, or 5 per cent, of these, paradoxical embolism had taken place, and in 3 of the 7 cases it was cerebral. And, as was brought out by Baumgartner and Abbott,[4] and observed in this collected series of cases, termination by brain abscess is especially liable to occur in ventricular septal defect, particularly so when there is dextroposition of the aorta, such as occurs in the "Tetralogy of Fallot," for then there is a more direct path for the transmission of the infected embolus.

The paradoxical brain abscess seems to occur somewhat more frequently in males than in females, the ratio in this series being 7 males to 4 females. The case with open foramen ovale and closed ventricular septum was in a female who had reached the age of fifty-seven; but the cases with ventricular septal defect all occurred at a comparatively early age. The age of the patients varied from nine and one-half to thirty years, the average being sixteen and one-half years. The source of the infected embolus cannot often be determined. In Case 5 it occurred after operation for acute appendicitis; in Case 7 it propably was due to the extraction of carious teeth; in the other cases the source was less definite. There does not appear to be any particular site of predilection in the brain for the occurrence of the abscess. Ours was the only case in which it occurred in the cerebellum.

The symptoms of paradoxical brain abscess do not differ essentially from the symptoms of brain abscess of any other source. The local signs and symptoms are the most important. Fever and a leucocytosis may be present. Eyeground examination and lumbar puncture may help in the diagnosis, depending on the location and size of the abscess.

SUMMARY

A congenital cardiac case presenting the combination known as the "Tetralogy of Fallot" and terminating with a paradoxical cerebellar abscess is reported, and a review from the literature of ten additional cases of paradoxical brain abscess is discussed. The sudden onset of cerebral symptoms in congenital cardiac cases, where interauricular or interventricular septal defects are suspected should lead one to consider a diagnosis of paradoxical brain abscess regardless of whether a septic focus can or cannot be demonstrated. On the other hand, the presence of an idiopathic brain abscess should lead one to suspect the possibility of the presence of a congenital cardiac anomaly.

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A LIGHT WEIGHT PORTABLE ELECTROCARDIOGRAPH

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THE clinical use of the electrocardiograph has been hampered by the excessive weight and bulk of the present so-called portable instruments, and also by the fact that the records taken by these portable instruments differ in many respects from the records taken with the standard string galvanometer. These defects of excessive weight and unsatisfactory recording are not inherent in portable electrocardiographs but can readily be eliminated by proper care in design and construction. The truth of this statement can be judged from the following description of a portable electrocardiograph which has been so constructed as to combine a maximum of serviceability with a minimum of inconvenience.

The characteristics of this instrument are as follows:

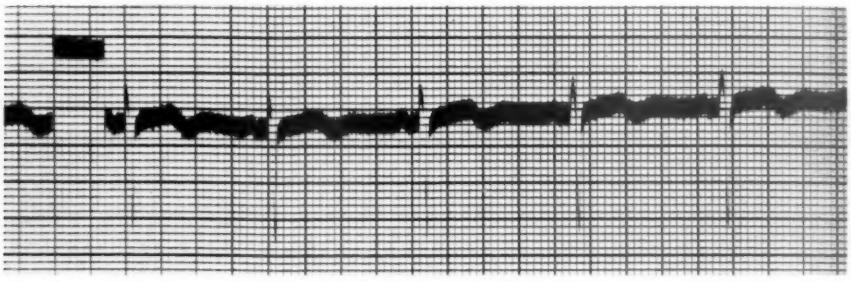

Fig. 1.

1. The total weight is forty-three pounds complete with batteries in two metal boxes which measure 7 by 9 by 11 and 7 by 9 by 18 inches respectively.

2. The record taken is of standard width (6 centimeters). It may be recorded on either film or paper. It is a replica of the record taken with the standard string galvanometer.

3. The instrument operates from its own batteries and is independent of the house current.

4. Durability and accuracy have been tested by frequent use for the past nineteen months.

Fig. 1 shows a record taken with this instrument.

The instrument operates by means of a three stage amplifier of simple construction, using standard tubes obtained in the open market. This amplifier has been especially designed to avoid distortion. The accurate amplification of the QRS complex is comparatively simple but the T-waves present special difficulties and special attention was paid to them in the design, with the result that the slowest

T-waves are amplified without appreciable distortion. This is indicated by the fact that if a constant voltage is applied to the input of the amplifier (see Fig. 1) the record shows a deflection which is practically horizontal for over one-fifth of a second.

The galvanometer, which is of the moving mirror type, has a natural period considerably shorter than 1/100 of a second. It is critically damped so that it can be very quick without overshooting under any conditions. This galvanometer reflects a very wide beam of light in the center of which is the shadow of a wire, thus duplicating the picture produced by a string galvanometer and permitting the use of a time marker of simple construction.

The apparatus is made mostly of duralumin and aluminum; no structural parts are made of wood. Thus it is rugged and rigid, and it cannot warp out of shape with changes of climate. A single electric motor operates the camera and the time marker. Its speed is accurately controlled by a special speed control which can be relied upon within a fraction of one per cent. The batteries used are Edison alkali batteries which have a long life and do not give off corrosive fumes.

The amplifier box contains the amplifier with its batteries and a control panel. This panel carries a main battery switch, a filament rheostat, a voltmeter indicating both high and low voltage, a lead selector, and a switch for applying a millivolt, used in standardizing the instrument. A three conductor cord goes to the patient, and a two conductor cord is plugged into the camera box.

The camera box contains the galvanometer and optical system, the film, the time marker, the driving mechanism and its storage battery, and a control panel. The control panel carries a jack to receive the amplifier plug, a knob to adjust the light beam position, a knob to adjust sensitivity, a switch to turn on the light and motor, and a periscope to view the heart action during operation.

In contrast to the string galvanometer the sensitivity of this electrocardiograph is not affected by skin resistance, and adjusting the sensitivity does not involve changing the speed of deflection. It can be operated in broad daylight.

It is to be emphasized that the light weight and other desirable characteristics of this instrument have been achieved solely through careful design and without the sacrifice of any essential parts. Its construction has involved great care to make the instrument not only portable but durable. And finally, the construction of this electrocardiograph has involved a far smaller financial outlay than would have been involved in the purchase of any of the available commercial electrocardiographs.

PORTABLE ELECTROCARDIOGRAPH GIVING DIRECT INK TRACINGS*

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MANY attempts have been made to record *mechanically* the action currents of the human heart by means of vacuum tube amplifiers. The difficulties are considerable, owing to the weakness and low frequency of the electrocardiac current.

Different types of amplifiers have been employed, but they have all proved insufficient to give a faithful record of the electrocardiograms.

The great clinical interest attached to this problem in medical practice induced us to make further attempts. By the use of the "ticker" we have been able to surmount the difficulties arising from the natural form of biological currents.

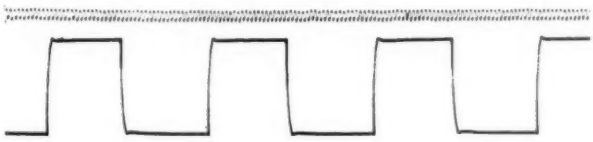

Fig. 1.—Record of a current of 1 millivolt. Timing 1/50 sec.

The ticker is an interrupter which has been used primarily in wireless telegraphy for the purpose of audition. Its application to the amplification of action currents has given us entire satisfaction.

The ticker in combination with an amplifier increases the intensity of action currents of the heart to such an extent as to set in motion a mechanical writing oscillograph. This oscillograph is an electrodynamic arrangement specially designed for this purpose by us. It consists essentially of a moving coil to which is attached a glass pen which records in ink the tracings on ruled paper. The ink supply is derived from a special container which prevents overflowing during transport.

The maximum range of the pen on the paper is 6 cm. and the maximum periodicity 100 per sec.

Special arrangements are provided to prevent overshock of the moving pen. The tracing in Fig. 1 shows the efficacy of this contrivance.

The amplifier and the oscillograph are under complete electrical and mechanical control. Figs. 2 and 3 effectively demonstrate this point, and show at the same time the similarity of the curves obtained by an optical standardized electrocardiograph (Boulitte) and our direct ink tracing method.

*From the Medical Clinic and Physical Laboratory, Geneva University, Switzerland.

At the same time these curves prove the accuracy of the electrocardiographic systems with amplifier, which are not inferior to the string galvanometers.

The sensibility of our electrocardiograph is regulated by the degree of amplification, whereas in the optical systems it is regulated by the tension of the string. The tension of the steel wires which support the moving coil in our electrodynamic oscillograph is constant, and this fact

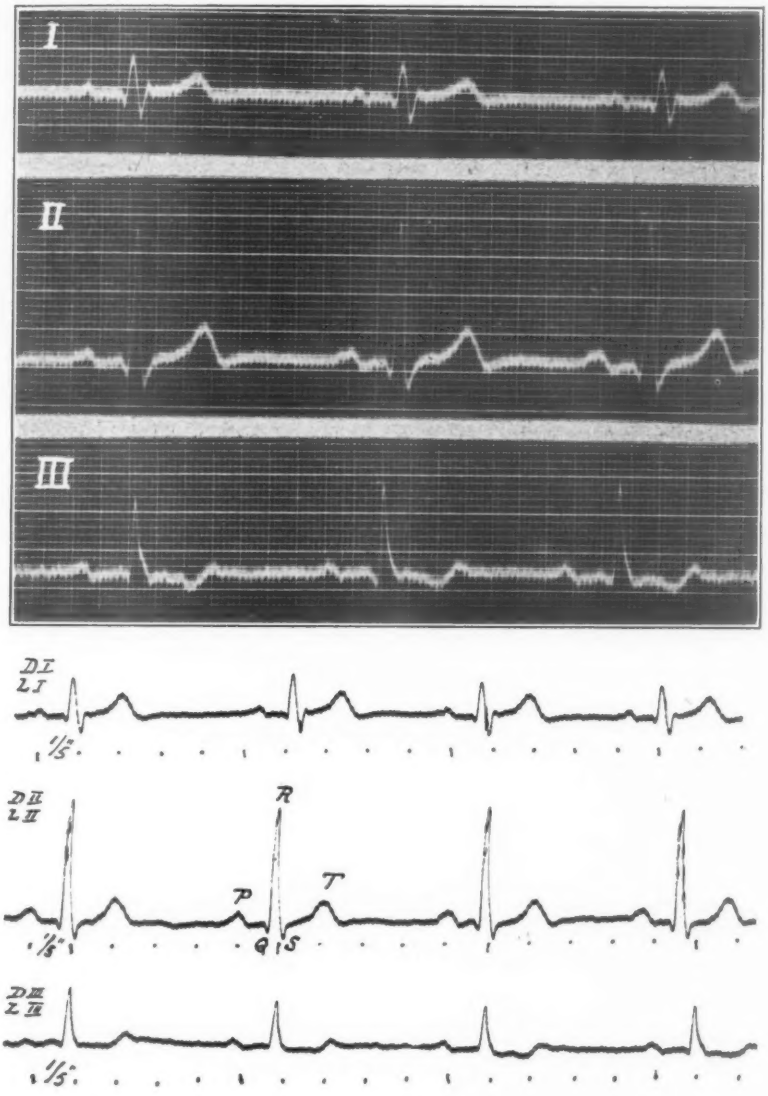

Fig. 2.—Normal case. The same electrocardiogram taken with an optical standardized apparatus (Boulitte) and the direct ink tracing electrocardiograph. (Natural size.)

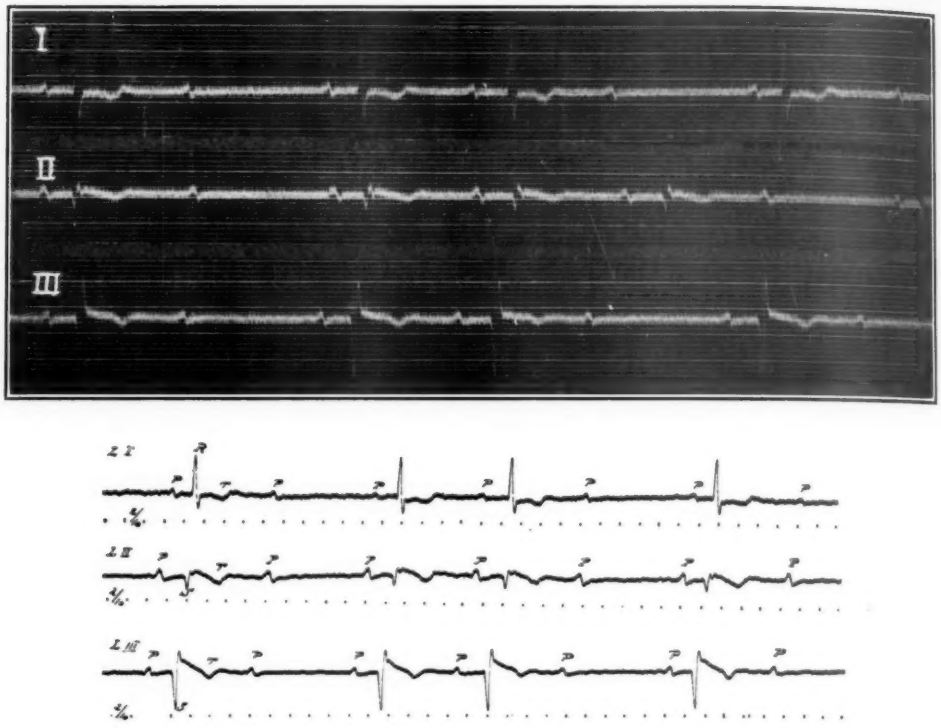

Fig. 3.—Myocardial infarction. Electrocardiograms taken at the same time with the optical method and the direct ink tracing apparatus (eight days after a severe attack). Auriculo-ventricular block, typical T-wave. (Reduced 1/3.)

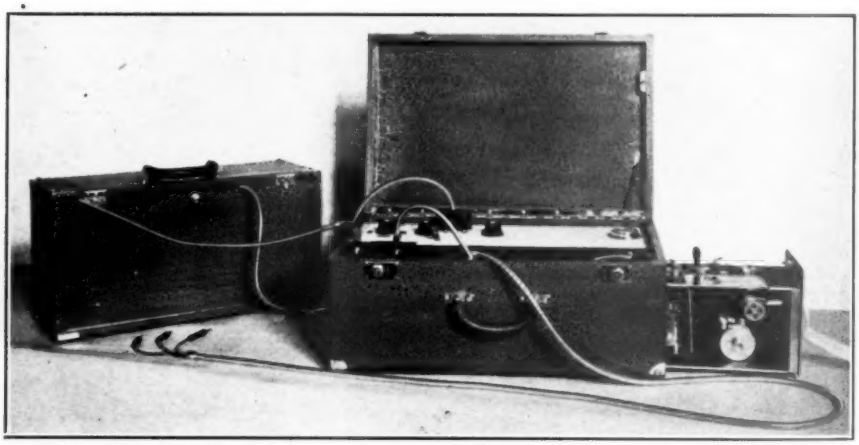

Fig. 4.—Portable electrocardiograph giving direct ink tracings. To the right one sees the oscillograph with its writing pen and the tracing issuing from the box.

guaranties the regularity of the curves independently of the resistance of the patient under examination.

In addition, Figs. 2 and 3 show that the ink tracings are not inferior to the photographic tracings in the precision of the waves or the neatness of the curve.

Our portable direct ink tracing electrocardiograph consists of the two neat cases whose total weight is 37 kilograms (Fig. 4). One case contains the accumulators and the other the electrocardiograph. By means of this apparatus the physician is able to obtain a complete electrocardiogram by the bedside of the patient in less than ten minutes.

Our object in presenting this apparatus to medical practitioners is twofold: first, the great simplicity in its application, and second, the advantage of having an exact tracing on the spot.

Department of Clinical Reports

SUBACUTE BACTERIAL *(STREPTOCOCCUS VIRIDANS)* ENDOCARDITIS AND ENDARTERITIS INVOLVING THE TRICUSPID VALVE AND THE PULMONARY ARTERY IN A UNIQUE CASE OF THE TETRALOGY OF FALLOT COMPLICATED BY CONGENITAL PULMONARY REGURGITATION

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THE termination of life in congenital cardiovascular disease by the complication of subacute bacterial *(Streptococcus viridans)* endocarditis and endarteritis is not rare. Maude Abbott[1] reports that among 555 cases of congenital cardiac disease analyzed by herself, 98 (or 17.6 per cent) presented such subacute bacterial invasion. Of 82 of these cases, 20 (or 23 per cent) were cyanotic patients with pulmonary stenosis, made up almost wholly, in all probability, of instances of the tetralogy of Fallot (pulmonary or infundibular stenosis, interventricular septal defect, dextroposition of the aorta, and right ventricular enlargement). An analysis from the opposite point of view by Abbott on the same page reports that among 84 patients with pulmonary stenosis and ventricular septal defect there were 25 cases (30 per cent) of acute or subacute endocarditis. Examples of the complication of subacute bacterial endocarditis and pulmonary endarteritis in the tetralogy of Fallot have been published by Abbott[3] and by Leadingham.[4] Involvement of the pulmonary artery by subacute bacterial infection has been discussed by a number of authors, including Mehlin[5] and Gordon and Perla;[6] the majority of such cases show patency of the ductus arteriosus.

We have obtained postmortem examinations in three of our private patients with the tetralogy of Fallot. The first two patients died of heart failure, complicated by cerebral symptoms, and of apoplexy respectively, at the ages of twenty-three and of sixty years. The third case, which forms the basis of the present report, was a young woman aged twenty-one years who died of subacute bacterial *(Streptococcus viridans)* infection involving the tricuspid valve and the pulmonary artery. Her heart was especially remarkable in that it showed in addition to the tetralogy of Fallot a congenital defect in the pulmonary valve itself, giving rise to pulmonary regurgitation, which lesion rendered the exact diagnosis clinically a difficult one. Congenital pulmonary regurgitation is very rare, having been noted only 12 times in Maude Abbott's series of 1000 cases,[2] but we ourselves happen to have

found the condition once before at postmortem examination,[7] and in that case the cusps were entirely missing.

The present case is reported, therefore, partly because of its rarity and party to throw light on the establishment of the correct diagnosis of pulmonary regurgitation in similar cases in the future.

CASE REPORT

W. H. M., a young woman student, aged twenty-one years, entered the Baker Memorial Hospital (Massachusetts General Hospital) on April 8, 1930, with a history of an acute illness of six weeks' duration. Trouble started with a "cold in the head" and fever. There was a coryza for a few days only but the fever persisted. Diarrhea came on after the head cold had subsided, and for several weeks there were three to four watery movements a day without pain and without blood or mucus in the stools. Gradually the diarrhea subsided but the stools were still abnormally frequent and loose on admission to the hospital. Her diet had consisted mostly of buttermilk for the first part of the illness but was changed later to a light mixed diet. For two weeks before entrance to the hospital a mild cough had existed.

Her past history revealed one important point. At the age of five years, in 1914, she had been examined by a pediatrician for some minor complaint and a harsh systolic murmur with palpable thrill had been found in the pulmonary valve area. In 1916 there were the same findings along with a suspicion of active endocarditis. She was sent to a children's heart hospital for a few weeks and discharged in good condition. At the age of nine years in 1918 she had her tonsils and adenoids removed. On repeated examinations, the last of which was in 1924, the heart is said to have shown what it did at first. After long continued observation over a period of ten years which included the finding of slight transient cyanosis on exertion, a final diagnosis of some kind of congenital heart disease was made by the pediatrician who had seen her first in 1914. The only infections in childhood were measles and chickenpox. There was no history of scarlet fever, rheumatic fever, diphtheria, or pneumonia.

The family history was good. Father, mother, one brother, and one sister were living and well.

Physical examination on April 8, 1930, on admission to the hospital, showed a young woman lying comfortably, flat in bed with slight but definite cyanosis of the lips and toes but little or no clubbing of either fingers or toes. There was no dyspnea. No petechial hemorrhages or nodules were found anywhere over the body. The heart showed slight enlargement, the apex impulse and left border of dullness being found in the fifth intercostal space 9.5 cm. to the left of the midsternal line and 1.5 cm. beyond the midclavicular line. The heart sounds were normal. There was a loud harsh systolic murmur heard best at the left of the upper sternum in the second and third interspaces and not well heard elsewhere over the precordium. It was attended by a palpable thrill. There was also heard a slight but definite blowing early diastolic murmur at the left of the upper sternum maximal in the second space. The two murmurs were not continuous but were distinctly separated from one another. The lungs were clear but showed harsh breath sounds throughout. The abdomen was normal. Liver and spleen were not palpable. There was no edema over back or legs.

At this time, before further study was made, the following comments were written in the record: "The diagnosis is very difficult. The positive findings indicate infection and heart disease. Whether these two are now related is the question to determine. The heart condition suggests strongly a very unusual pulmonary valve

lesion, perhaps congenital (that is, pulmonary stenosis), perhaps acquired (pulmonary stenosis and regurgitation) or perhaps combined (that is, subacute bacterial endocarditis complicating a congenital lesion). There does not seem to be definite evidence of mitral or aortic disease. *Treatment.* Symptomatic therapy and continued study as to the cause of the fever and diagnosis as to heart lesion. *Probable diagnosis:* Subacute bacterial endocarditis involving the pulmonary valve, perhaps superimposed on congenital pulmonary stenosis. Bacterial endocarditis complicating congenital patency of the ductus arteriosus is the other possibility."

On April 10, 1930, the following note was made: "The character of the murmurs in the pulmonary valve area is not typical of patent ductus arteriosus. The time of the loudest murmur and thrill is systolic. Pulse is not Corrigan."

On April 11, "Fluoroscopic examination shows marked prominence and bulging in the region of the pulmonary artery with vigorous pulsation, appearing to be a Corrigan pulse in the pulmonary artery. There is also well marked pulsation of the lung hiluses. Heart somewhat enlarged.

"Electrocardiogram shows normal rhythm, rate 105, slight to moderate right axis deviation, but not typical of marked congenital pulmonary stenosis."

On April 14, "The report of a positive blood culture confirms the diagnosis of bacterial endocarditis."

On April 18, "Spleen not palpable. Condition shows no appreciable change. Murmurs as before. No petechial hemorrhages or evidence of embolism."

Dr. Maude E. Abbott saw this patient in consultation on April 18 and made the following note: "Tentative diagnosis—Patent ductus arteriosus with dilatation of the pulmonary artery and infective pulmonary endarteritis, beginning around the pulmonary orifice of the ductus arteriosus, probably extending down to and involving the pulmonary valve with pulmonary insufficiency from destruction of the cusps." Our own note that same day follows: "This diagnosis by Dr. Maude Abbott is very logical and seems likely to be correct. There is no specific therapy and the prognosis is bad. It is probably a matter of a few weeks."

On April 26 she was discharged from the hospital with the following note: "There has been no appreciable change in her condition since entrance except for a moderate increase in the secondary anemia and the development of slight clubbing of the fingers. It is probable that there will be some weeks or even months before the termination of this illness. The prognosis is hopeless." The diagnosis on discharge was: "Congenital patency of the ductus arteriosus with subacute bacterial endocarditis and endarteritis, involving the ductus arteriosus, pulmonary artery, and pulmonary valve. *Streptococcus viridans* is the causative agent of the endocarditis."

During the next two months this patient gradually failed at home and died on June 25, 1930, having had "acute meningeal symptoms for the last day or two." The known duration of the illness was four months.

LABORATORY DATA DURING HOSPITAL STAY

The temperature chart showed constant fever, ranging from 99° to 104.5°, with daily swings from the lowest figures in the morning to the highest in the evening.

Urine, April 8, 1930. Normal, acid, specific gravity 1.014, very slight trace of albumin, no sugar, bile or pus. Sediment: occasional squamous cells, occasional leucocytes, few bacteria, rare red blood cells.

Blood counts and smears, April 9, 1930. White count 19,500, red count 4,450,000, Hgb. 70 per cent. Differential leucocyte count: 87 per cent polymorphonuclears, 8 per cent large lymphocytes, 4 per cent small lymphocytes, 1 per cent large mononuclears. Moderate achromia and marked polychromatophilia of red blood corpuscles with variation in size and shape. Platelets decreased.

April 18, 1930. White count 12,500, red count 3,650,000, Hgb. 55 per cent. Polymorphonuclears 83 per cent, small lymphocytes 13 per cent, mononuclears 2 per cent, unclassified 2 per cent. Slight achromia and slight variation in shape of red cells. Platelets slightly decreased in number.

Blood cultures April 9, 1930. Both flasks showed *Streptococcus viridans.*

April 15, 1930. 1. *Streptococcus viridans.* 2. *Streptococcus viridans.*

April 16, 1930. Both blood cultures showed *Streptococcus viridans.*

Electrocardiogram, April 9, 1930, showed normal rhythm, rate 105, and slight right axis deviation.

POSTMORTEM EXAMINATION, JUNE 25, 1930

The examination was performed eight hours after death, the thorax only being opened. There were slight cyanosis and a moderate clubbing of the fingers.

The lungs were moderately congested, and on the left the lower lobe was compressed and atelectatic, being displaced laterally and upward by an enlarged heart. The right lower lobe contained several small wedge-shaped areas, having the typical appearance of infarcts.

The heart weighed 540 grams. It was decidedly abnormal in shape, due in part to a dilatation of the right auricle, but principally to an hypertrophy of the right ventricle, producing a blunting of the apex, the whole organ assuming a roughly rectangular shape. The heart before removal from the chest was noted to lie almost at right angles to the midline.

The apex of the heart was made up practically entirely of right ventricle. Three-fourths of the anterior surface consisted of the right ventricle and one fourth of the left. The posterior surface was made up two-thirds of right ventricle and one-third of left ventricle.

The length of the heart from the base of the aorta to the apex was 11.5 cm., and from the superior vena caval opening to the apex, 15 cm. The anteroposterior thickness of the ventricles was 8 cm.

The pericardium was normal.

The chambers of the heart and the great vessels were in normal relationship. The venae cavae were normal and emptied into the right auricle. The latter was dilated to twice the normal size and its wall was slightly thickened. The foramen ovale was closed.

The tricuspid valve was slightly thickened, and on its cusps were many vegetations, most numerous on the posterior cusp where they consisted of long pedunculated grayish white masses, hanging down into the ventricular cavity, some measuring as much as 2.5 cm. in length. On the medial and anterior cusps there were a few shaggy vegetations only a few millimeters in length. The circumference of the valve was estimated to be 10 cm.

The right ventricle was dilated and markedly hypertrophied, being considerably larger than the left. The wall measured 12 mm. in thickness in the main cavity, thinning down to about 4 mm. in the pulmonary infundibulum. The muscle was of normal color and consistency. The infundibulum was 3 cm. in length and 22 mm. in diameter at its origin, narrowing gradually as it approached the pulmonary valve.

The pulmonary valve measured 15 mm. in diameter and was composed of two cusps, leaving when closed, a narrow slitlike opening 14×3 mm. in size. One cusp extended the full diameter of the artery, and behind its free edge the shallow cup was divided into unequal portions by a narrow fibrous ridge extending from the free margin to the wall of the artery. The opposing cusp did not completely cover the other half of the artery, but left a small area 2 to 3 mm. in width on the anterior surface. This latter smaller cusp was more than twice as deep as the opposite one. There were no vegetations on the pulmonary valve.

The pulmonary artery was markedly dilated from the level of the valve to its first branches. About 3 cm. above the valve, it measured 9 cm. in circumference. On the external surface of the artery, beginning just above the level of the valve, and over an area about 4 cm. square, the wall was reddened, injected, roughened, and seemingly thinner than the remainder. On the intimal surface, corresponding to this area, the wall was covered with numerous shaggy vegetations, similar in appearance to those found on the tricuspid valve. The remainder of the pulmonary artery was negative except for the dilatation mentioned above. The ductus arteriosus was closed.

The left auricle was of normal size and thickness and contained no thrombi.

The mitral valve measured 8 cm. in circumference and except for a slight thickening of the cusps was normal in appearance. There were no vegetations on the valve.

The left ventricle, comprising less than one-half the bulk of the heart, was of normal size and not dilated. The muscular wall measured 11 mm. in thickness. The papillary muscles were not abnormal.

There was a defect in the interventricular septum measuring 3×1.5 cm., 75 cm. above the apex of the left ventricle, and immediately below the dextroposed aortic valve.

The channel leading to the aorta from the left ventricle measured about 2.5×1 cm., one side consisting of the aortic cusp of the mitral valve. In the right ventricle, the channel leading to the aortic valve was more direct and slightly larger, measuring 3×1.5 cm., at the uppermost portion of the septum where these two channels joined. At the level of the defect, the septum varied from 6 to 8 mm. in thickness.

The aortic valve was situated more over the right than over the left ventricle. Its three cusps showed some variation in size, there being one large and two small cusps. The large cusp comprised about one-half the valvular surface, the remainder of the area being covered about equally by the two smaller cusps. At the level of the valve, the aorta measured 27 mm. in diameter.

The aortic arch was smooth and of normal size. There was no coaretation.

The coronary arteries were normal in size, and the walls were not thickened or sclerosed. The right coronary artery took its origin behind the large aortic cusp, and supplied practically the entire right ventricle. The left coronary artery branched normally and supplied the remainder of the heart.

Pathological Diagnoses: Congenital anomalies of the heart, (interventricular septal defect, pulmonary stenosis and regurgitation, dextroposition of the aorta, hypertrophy of the right ventricle), and bacterial *(Streptococcus viridans)* endocarditis of the tricuspid valve and of the pulmonary artery.

DISCUSSION

That subacute bacterial endocarditis complicating congenital cardiovascular disease was present in this case was evident after a brief clinical study, but the failure to make an exact diagnosis of the structural defects was due to several reasons. In the first place the great rarity of congenital pulmonary regurgitation made such a diagnosis improbable, the diastolic murmur being thought at first to be the result of secondary involvement of the pulmonary valve by the infection and later to be a part of the manifestation of patency of the ductus arteriosus even though unusual in timing (that is, the murmur was not continuous). The marked prominence of the pulmonary artery on x-ray examination was at the time attributed most readily to patency of the ductus arteriosus,

without the realization that long continued pulmonary regurgitaion would be associated with pulmonary artery dilatation. The marked water-hammer pulse in the pulmonary artery as seen fluoroscopically was not given its correct significance as due to pulmonary regurgitation; patency of the ductus arteriosus does not produce so marked a pulmonary arterial pulsation as we found in this case. The second chief reason for our missing the exact diagnosis of structural defects in this case was the extraordinary paucity of evidence for the tetralogy of Fallot; the cyanosis was very slight, clubbing of the fingers was not apparent at all at first, and the blood showed a secondary anemia (due to the infection) rather than a polycythemia. Experience in this case should prove helpful in future cases.

SUMMARY

A very unusual case is reported of subacute bacterial *(Streptococcus viridans)* endocarditis and endarteritis involving the tricupid valve and the pulmonary artery in a young woman twenty-one years old who showed the tetralogy of Fallot complicated by congenital pulmonary regurgitation.

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POSTURAL HYPOTENSION WITH TACHYCARDIA. A CASE REPORT*

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BRADBURY and Eggleston[1] in 1925 reported their observations of three patients whose blood pressures regularly fell below the critical level on standing in the erect position for a few minutes, and in their report they used the term "postural hypotension" to represent the particular condition described. Since 1925, several other cases of postural hypotension have been reported.[2, 3, 4] The marked features of the first reported cases were: critical fall of blood pressure with change from the horizontal to the upright position; slow pulse rate; incapacity to perspire; lowered basal metabolism; and signs of indefinite changes in the nervous system. Other features present in some, but not in all, of the reported cases were: chronic diarrhea; greater excretion of urine by night than by day; loss of sexual power; false appearance of youth, and secondary anemia.

In reviewing the reported cases of postural hypotension, it is observed that the essential and characteristic features common to all were: (1) the falling of the blood pressure below the critical level with the assumption of the upright position, and (2) the physical and psychical symptoms which were direct results of cerebral and cerebellar anemia. The other phenomena, not common to all the cases, were evidently individual or incidental.

Another case of marked postural hypotension is here presented. In this case tachycardia and abnormal changes in the electrocardiogram accompanied the fall in blood pressure with the change from the horizontal to the upright position.

CASE REPORT

The patient, an electrician, thirty years of age, came under observation on November 30, 1931. He complained of marked dizziness, blurring of vision and faintness on sitting, standing or walking, sensations as of abnormal heart action, frequent nausea and distress in the upper abdomen, soreness of the lower abdomen, chronic diarrhea, and numbness and coldness of the hands and feet. He stated that all of these conditions had continued for several years and that for the past three years they had entirely incapacitated him from earning a livelihood.

The patient was kept under observation until December 29. The following notes are taken from the case records:

He states that he was a strong and healthy child and youth prior to entering the U. S. Army early in May, 1917, and that he had no sickness or injury of conse-

*From the U. S. Veterans' Administration Diagnostic Center.
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quence in the army service until about October 18, 1918, when he was "blown up" by a shell explosion, and, at the same time, was overcome by poison gas. He has no clear memory of what happened, and he thinks that he was unconscious for a short time. When he came to himself in a first aid station, he found that he had several slight and superficial wounds, that he had severe throat and lung irritation from the gas, and that he was very much upset nervously. He was returned to the United States as a "casual" and was continually under treatment in army hospitals until the follwing June. His weight early in 1918 had been 195 pounds. His weight in June, 1919, was 145 pounds. Since 1919 his weight has varied between 130 and 145 pounds.

From 1919 to 1928 he was greatly handicapped by dizziness and faintness when he was walking or standing, though he was able to hold a position for some years as resident operator of an electric power substation. He explains that in that position no strenuous exertion was required and that he was permitted to rest most of the time.

Since 1928 he has not been able to do any work. For this time he has not only been dizzy and faint when sitting, standing or walking, but he has also had much abdominal distress and an almost constant watery diarrhea. The abdominal distress has subsided and the stools have been formed only when he has rested in bed for several days. He has observed that his pulse has been slow while resting in bed, sometimes as slow as 40 per minute; but on rising from the bed his pulse has immediately become rapid. His wife has counted his pulse while standing and has reported that it is sometimes as fast as 160 per minute. He has often had "queer sensations" in his heart as though its action were irregular. When he has been walking, standing, or sitting for a time and has felt himself becoming dizzy and faint and his sight becoming blurred, he has learned to avoid serious injuries by immediately lying down or sitting down and bending forward with his head between his knees. On rising quickly from bed to a sitting position, he has often lost consciousness for a moment and has fallen to the floor and injured himself. Every time he has fallen he has regained consciousness on the instant that his head has become low.

He gives no history of having had rheumatic fever, chorea, scarlet fever, diphtheria, malaria, typhoid fever, pneumonia or tuberculosis. He has been subject to frequent chest pains, chronic cough, shortness of breath with exertion, and occasional mild asthmatic attacks since receiving the poison gas injuries in 1918. A few times he has raised slightly blood-streaked sputum, though he has never had a frank hemoptysis. He thinks that he perspires quite normally, and he has not observed that his general symptoms have been any more pronounced in the summer than in the winter. He has had no kidney or urinary trouble, to his knowledge. He has had no excessive thirst, and ordinarily he has had no nocturia. He habitually sleeps with a low pillow or with no pillow at all, and he has not been troubled with night dyspnea except occasional attacks of bronchial asthma. At various times he has observed that his feet and ankles have become swollen when walking, standing or sitting more than usual. For years his hands and feet have nearly always been cold and damp, and frequently his lower arm and leg become numb when he lies on one side.

His appetite is always poor. He has seldom vomited, though he is very frequently nauseated after eating. In recent years he has had much midepigastric distress, especially after meals. He has found these troubles to be least annoying when his food has consisted chiefly of milk and when he has continued much of the time in bed. Alkaline powders give almost immediate temporary relief from the epigastric distress, as does, also, the taking of milk during attacks. He has never been

jaundiced. He takes cream and other fats with no increase of the abdominal symptoms.

From 1918 to 1928 he was subject to attacks of diarrhea at intervals of a few months, and the attacks would usually last several weeks. For the past three years he has had two or three watery stools nearly every day. In recent years he has also had a lower abdominal distress and soreness, which he localizes as in the mid-area of the lower abdomen. He states that this distress has been constant rather than colicky and that it has been increased rather than decreased by the evacuation of the bowels. He had a left inguinal herniotomy in 1923 but has had no other abdominal operation.

He has never used much liquor, and none at all in recent years. He denies ever having used drugs, and there is nothing in his records or in his personal appearance that is suggestive of drug addiction. He smokes tobacco very moderately. He denies ever having had a venereal infection.

Family History.—The patient's father died of Rocky Mountain fever at sixty-five years of age. The mother is living and quite well at sixty-five years. He has had one brother and three sisters, and they are all living and well. He was married in 1926. His wife is well and has had two pregnancies by the marriage, each pregnancy resulting in a normal child, now living and well.

Physical Examination.—The patient is a very slender, poorly nourished and frail appearing white man, 73.5 inches tall and weighing 141.75 pounds, who has the appearance of his stated age of thirty-two years. He seems to be mentally alert, but he has a drawn and careworn expression of countenance. All his movements are slow and guarded. He has a very pronounced upper dorsal stoop, and he appears to be unable to stand erect, though he stands steadily with eyes closed. There are no notable tremors. The patellar reflexes are active.

His face and ears are very pale as he sits or stands, but their color becomes quite good almost immediately when he lies down. His hands and feet are moderately cyanotic when dependent, but they are of normal color when at the level of the body, and they retain their normal color after elevation for several minutes. His fingers are remarkably long and slender, and the finger nails are small, smooth and well shaped. His hands and feet are cold and damp. His skin is soft and quite free from lesions. There is an operative scar in the left inguinal area. No other gross scars of operation or injury are observed. There are no varicosities, and no definite edema is demonstrated.

His hair is thick, light brown, straight and rather coarse and oily. His face is symmetrical. He has no exophthalmos. The pupils are quite large. They are equal and regular, and they react to light, accommodation and convergence. The veins of the fundi are notably smaller when he is in the upright position than when he is in the horizontal position. The palpebral conjunctiva and the mucous membrane of the lips are very pale when he is upright. The thyroid gland is not palpable. The lateral cervical and the epitrochlear lymph nodes are just palpable. Other superficial lymph nodes are not remarkable.

Summary of Further Observations.—No definite evidences of disease were found in the upper respiratory tract, and the physical and roentgen-ray findings of the lungs were essentially negative. The roentgen-ray films showed total lack of calcification in the costal cartilages and a peculiar rarefaction of bone in the upper margins of the anterior ends of the third, fourth and fifth ribs on either side. The heart was shown to be rather small and centrally placed. A prominent, clearly outlined, oval shadow was observed in the position of the pulmonary artery. The significance of this shadow was not evident.

The visible and palpable cardiac impulses were slight, in the fifth interspace and eight centimeters from the midsternal line. No precordial thrills were palpable.

There was no abnormal precordial tenderness. The heart sounds were of normal character. Usually the rhythm was found to be regular, though at various times a rather marked sinus arrhythmia of the respiratory type was observed. A few times pulse irregularities were observed, the character of which was not definitely determined.

Most extraordinary changes in the pulse rate were produced simply by changing from the horizontal to the sitting position, and from the sitting position to the standing position. These changes in pulse rate were instant with the changes in position. The pulse rate regularly doubled with the change from lying down to standing, and this increase was approximately halved in the sitting position. This ratio of change in rate was fairly constant; when the pulse rate was 80 while lying in bed, it was found to be about 120 immediately when he sat up and 160 when he stood up; when the rate was 60 in bed, it was found to be approximately 120 when he rose to his feet. With the change from the upright to the horiztonal position, the pulse rate was almost instantly halved.

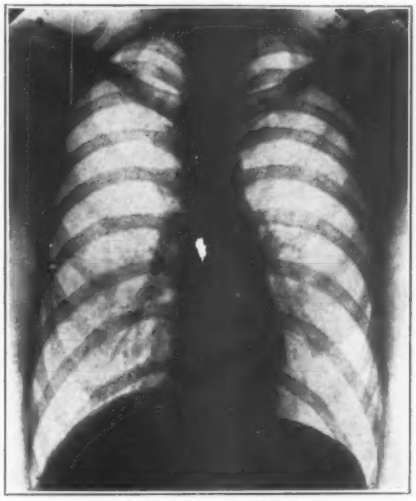

Fig. 1.

Even more remarkable than the variations of the pulse rate were the changes in the blood pressure readings with the changes in posture. With the patient lying down, the blood pressure readings were systolic 122 and diastolic 90 mm. With the patient sitting and with the sphygmomanometer cuff at approximately the level of his heart, the systolic reading was 115 and the diastolic 90 mm.; but when the arm was lifted so that the cuff was at approximately the level of his brain, the systolic reading was 80 and the diastolic 62 mm. With the patient standing and his arm at his side, the systolic reading was 90 and the diastolic 80 mm.; but when the arm was raised so that the cuff was at the level of his brain, the systolic pressure was 60 mm. and no definite diastolic reading could be made. Blood pressure readings in these various positions were repeated from time to time during the patient's stay in the hospital. The basic readings were variable, but, with the patient standing and with the cuff at the level of the brain, the systolic pressure was repeatedly read at 60 mm. On one occasion the systolic pressure at the brain level was read at 60 mm., and the patient was then required to stand, leaning against the wall, for ten minutes. The systolic reading at the brain level was then 50

mm. The patient asserted that he was not unusually faint, though his face and ears appeared to be quite bloodless.

The abdomen was long, flat and almost devoid of subcutaneous fat, but the musculature was fairly good, and there were no notable evidences of sagging of the abdominal viscera. No abnormalities could be palpated in the abdomen, though two areas of tenderness were located, one in the midepigastrium, the other midway between the umbilicus and the symphysis pubis. Firm supporting pressure on the lower abdomen, while the patient was in the upright position, caused him to complain bitterly of increased abdominal discomfort. His pulse continued weak and rapid while this pressure was applied, and some irregularity of the rhythm was noted, but the mechanism of the irregularity was not definitely determined.

Because of the report[2] of very satisfactory results in postural hypotension with the oral administration of ephedrine, it was hoped that similar results might be

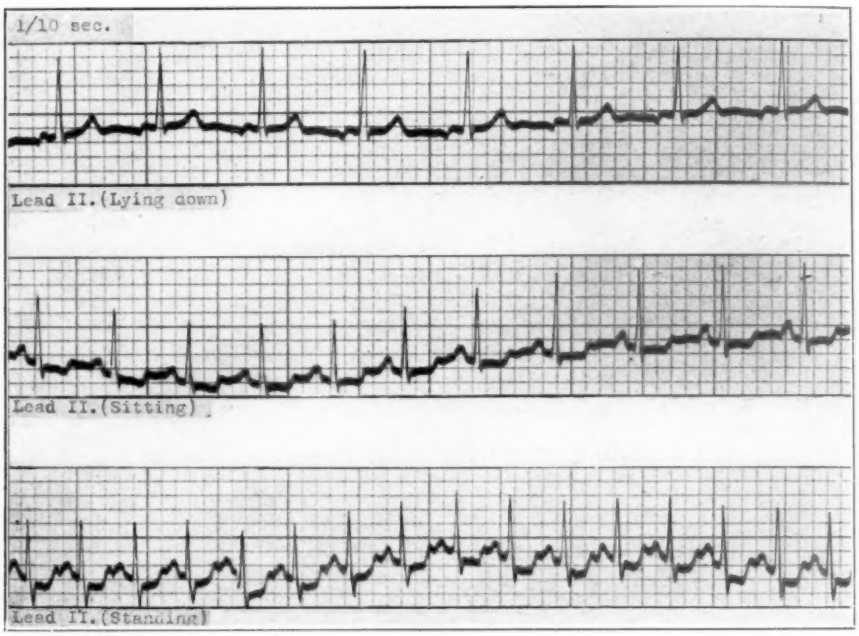

Fig. 2.—Case of postural hypotension.

obtained in this case. On December 19 the following notation was made on the patient's ward record: "At 12:45 P.M. the patient was given a ¾ grain capsule of ephedrine sulphate, as a therapeutic test, in an attempt to raise his blood pressure. He complained of a chilly feeling shortly after taking the ephedrine. At 2:30 P.M. he reported that he had experienced a rather severe chill during the previous half hour, that his head had felt queer and that he had had sensations as of irregular heart action. However, he stated that those annoying symptoms were nearly passed. He was then lying quietly in bed and his pulse was quite regular and of good quality, at the rate of 60 per minute. He was asked to stand up by the bed. Immediately on standing, his pulse became very weak, its rate rose to 120 and his face became very pale. After standing for about a minute, he suddenly slumped to the floor. He no doubt would have injured himself had he been alone. Instantly, as his head became low, he recovered consciousness. After a few minutes

he was assisted to the sitting posture on the edge of the bed. Again his pulse became rapid and weak, and in a few moments he fell over in another faint. He recovered consciousness again immediately as his head went low, and almost instantly his pulse was slow and of good volume. These are the first times the patient has fainted since admission to the hospital. Further ephedrine medication is not deemed advisable.''

Laboratory Data.—Blood: hemoglobin, 90 per cent; total red blood cells, 4,800,000 per c.mm.; total white cells, 7400; polymorphonuclears, 63 per cent; small mononuclears, 31 per cent; large mononuclears, 3 per cent; transitionals, 1 per cent; eosinophils, 1 per cent; basophils, 1 per cent. Red cells of normal appearance. Blood Wassermann reaction negative. Blood sugar: fasting, 125 mg.; one and a half hours after tolerance meal of 100 grams of glucose, 130 mg. Icterus index, 7. Van den Bergh tests: direct, negative; indirect, normal. Four basal metabolism estimates averaged −8.

Gastric analysis of test meal: Total acidity, 46; HCl 34; bile, +2; occult blood, +1; gastric mucus, +1. Eleven sputum reports were negative. Urinalysis reports were negative. Of 10 feces, 6 contained occult blood. No parasites or ova were found.

The report of the roentgen-ray examination of the gastrointestinal tract reads in part: ''The stomach outlines readily and shows no defects. The duodenal bulb fills well. It shows a slight irregularity on its greater curvature near its apex. This is not constant and is of doubtful significance.'' The stomach was of the fish-hook type. The transverse colon was near the level of the iliac crests. The films at six hours, twenty-four hours and forty-eight hours revealed no definite abnormalities.

COMMENT

The three electrocardiograms here presented were taken within a few minutes. The first was taken with the patient lying in bed. For the second, he was asked to sit upon the side of the bed, the electrodes being still attached to his wrists and ankle. After about a half minute, the time required for the beam of light to become adjusted, the second record was taken. Then the patient was required to stand by the bedside while the third record was taken. The order of procedure was then reversed, and electrocardiograms were taken immediately on sitting down and on lying down. Only one of the two series is here presented, as they were found to be identical.

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3. Riecker, H. H., and Upjohn, E. G.: Postural Hypotension, Am. Heart J. **6**: 225, 1930.
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Department of Reviews and Abstracts

Selected Abstracts

McMillan, Thomas M., and Bellet, Samuel: Auricular Flutter: Some of Its Clinical Manifestations and Its Treatment Based on a Study of 65 Cases. Am. J. M. Sc. **184**: 33, 1932.

A series of 65 cases of auricular flutter is reported, 12 of which were classed as paroxysmal and 43 as established flutter. Seven were due to drugs and 3 received no treatment and were therefore unclassified. The effects of digitalis upon the auricular rate are discussed. It is believed that the results of treatment of the disturbance were quite successful. Of the entire 65 cases a return to normal rhythm was secured in 66.1 per cent. Considering only the cases of established flutter, normal rhythm was successfully restored in 74.4 per cent of the cases.

The method of choice in this series of cases was the use of digitalis until fibrillation was established. Digitalis was then discontinued and unless a normal rhythm had spontaneously returned within a week, the administration of quinidin was begun. The latter drug was used alone after digitalis had either failed to bring on fibrillation or had brought on certain toxic manifestations which rendered a further use of digitalis inadvisable.

Landis, Eugene M., Jonas, L., Angevine, M., and Erb, W.: The Passage of Fluid and Protein Through the Human Capillary Wall During Venous Congestion. J. Clin. Invest. **11**: 717, 1932.

It was the purpose of these studies to measure the effect of graded venous congestion on the movement of fluid from the blood to the tissue spaces. Blood samples removed from the arm veins were compared in order to measure the filtration of fluid resulting from the increased venous pressure. The loss of protein through the capillary wall was estimated at venous pressures of 80, 60 and 40 mm. Hg. Comparison of hemoglobin readings and red cell counts showed that during venous congestion the fluid is lost chiefly from the plasma. The loss of fluid could be detected at venous pressures as low as 20 mm. Hg and amounted to between 0.0 and 2.3 c.c. per 100 c.c. of blood. The amount of fluid lost from the blood was conspicuously greater at higher venous pressures; venous congestion of 80 mm. Hg filtered as much as 19.5 c.c. per 100 c.c. of blood.

At a venous pressure of 80 mm. Hg protein was lost from the blood plasma in an amount indicating that the capillary filtrate contained an average of 1.5 per cent of protein. At a venous pressure of 60 mm. Hg very little protein loss could be detected and the capillary filtrate contained an average of 0.3 per cent protein.

Two cases of edema are described in which fluid was collected during venous congestion. The protein content of the edema fluid was 0.39 and 0.09 per cent, indicating that the capillary wall retained approximately 95 per cent of the plasma protein.

Dameshek, William, and Loman, Julius: Direct Intra-Arterial Blood Pressure Readings in Man. Am. J. Physiol. **101**: 140, 1932.

The intra-arterial pressure in about 50 individuals was determined by a direct method which was compared with the usual indirect ones. This direct method con-

sisted of introducing a 20-gauge needle into the artery, usually the brachial, and connecting it through a 3-way stopcock with a syringe and an aneroid sphygmomanometer. The method used was subjected to a large series of experiments to determine its reliability. It was finally determined that the greater the pulse pressure the less accurate was the technic used, that a mean rather than a systolic pressure was determined.

The pressure within the common carotid, femoral and brachial arteries was compared in eight cases. The brachial artery pressure was slightly higher than that of either the carotid or femoral arteries. The apparent discrepancy between indirect and direct blood pressure readings in arteriosclerosis was concluded to be due to the high pulse pressure which is present in that condition. The indirect method of sphygmomanometry appears to be accurate even in the presence of marked arteriosclerosis.

Reid, William D., and Levene, George: Roentgenologic Consideration of Mitral Heart Disease. New Eng. J. Med. **206**: 1026, 1932.

While early mitral disease is often difficult of recognition clinically, it frequently may be identified by careful roentgen examination. The authors have recently described a method of recording the various measurements of the heart and have stated that the ratio that exists between the size of the auricles and ventricles may be of importance in diagnosis. In mitral stenosis an altered relationship of auricular and ventricular areas is a typical and diagnostic roentgenographic sign. They believe that the high index of this ratio is the most important single factor in the early roentgenographic identification of this condition. Mitral insufficiency can be demonstrated by roentgen examination and the organic and functional forms differentiated.

Roentgen examination affords accurate information concerning the course of mitral disease under treatment.

Lichtman, S. S., and Gross, Louis: Streptococci in the Blood in Rheumatic Fever, Rheumatoid Arthritis and Other Diseases. Arch. Int. Med. **49**: 1078, 1932.

A study of 5,233 consecutive blood cultures in a general hospital shows that with adequately sensitive methods an incidence of nonhemolytic streptococcemia between 4 and 15.5 per cent with an average of 6 per cent occurs in at least nine diseases. That is, acute rheumatic fever with polyarthritis, chronic rheumatic cardiovalvular disease, rheumatoid arthritis, aplastic anemia, pernicious anemia, leucemia, colitis, meningococcus meningitis and pyelitis and pyelonephritis. On the basis of the incidence of the "transient" streptococcemia alone, these organisms cannot justifiably be considered as the causative agents of these diseases.

Styron, Norma C., and Spicer, Sophie: Dissociation of Streptococcus Cardio-Arthritidis. J. Infect. Dis. **50**: 490, 1932.

The work on microbic dissociation reported in this paper has been done with the R 1 and R 9 strains of S. cardio-arthritidis isolated by Small from blood cultures of patients with rheumatic fever. Fifty cubic centimeter volumes of phosphate broth with or without dextrose, enriched with 2, 5 and 10 per cent concentrations of peptone support dissociation. The percentage of peptone most favorable to the process is 5. Strain R 9 was much more unstable than strain R 1. As a result of dissociation, two culture types have been obtained for each strain. As far as tested, no qualitative difference in carbohydrate fermentation, in action on milk and in production of indol has been demonstrated. No tendency to form methemoglobin was evidenced in any of the types.

Intradermal skin tests in rabbits to test the allergic capacity of the two types

resulted in lesions having distinct characters. The antigenic relationship of the two strains have also been investigated. It was found that cultivation in fluid mediums readily caused one of the strains, R 9 to show a reversion of the atypical forms to the more typical ones. A partial change from typical to atypical in the same strain was sometimes accomplished but it was very infrequent.

Dikar, Lewis: Acute Bacterial Endocarditis Due to Bacterium Acidi-Lactici. Arch. Int. Med. **49**: 788, 1932.

Two cases of Bacterium acidi-lactici endocarditis closely resembling cases of Bacterium coli are reported because of their rarity. It is believed that more complete bacteriologic studies of cases of bacterium coli sepsis will undoubtedly bring to light more examples of Bacterium acidi-lactici infections. The coincidence of aortic valve involvement in two cases of Bacterium acidi-lactici endocarditis and in two cases of Bacterium coli endocarditis is pointed out.

Don, C. S. D., Grant, R. T., and Camp, P. D.: A Case of Complete Heart Block With Varying Ventricular Complexes. Heart **16**: 145, 1932.

A case of complete heart block is described in which the ventricular complexes vary in form. Pathological examination showed that the bifurcation of the A-V bundle was destroyed by disease; the main bundle was separated from its branches and the right branch from the left. It is pointed out that these findings are similar to the results of experiments on dogs carried out by Wilson and Herrman in which both bundle branches were cut. Electrocardiograms from this patient are like those of the dogs and show changes in the ventricular complexes which can be interpreted as due to a shifting of the pacemaker from one ventricle to the other with transitional forms showing that two pacemakers were competing for the ventricular rhythm. The intravenous injection of atropin temporarily abolished the variation in the ventricular complexes which assumed an intermediate form.

Grant, R. T., and Camp, P. D.: A Case of Complete Heart Block Due to an Arterial Angioma. Heart **16**: 137, 1932.

A case is described of complete heart block developing in an adult and due to destruction of the terminal portion and bifurcation of the A-V bundle by an arterial angioma. At autopsy the examination of the heart revealed an angioma similar to those which may be found elsewhere in the body. A clinical history in the present case indicated the growth of the tumor during the last few years of life so as to involve the conducting system of the heart.

Gil, Urbano Gonzalez: The T Wave of the Electrocardiogram. Its Pathological Interpretation and Prognostic Value. Arch. de Cardio. Y. Hemato. **13**: 187, 1932.

The author has attempted to decide in the present work the importance of the different causes which give place to an inversion of the T-wave in order to judge the prognostic value of this electrocardiographic alteration. At the same time, it is interesting to know the precise difference between the pure inversions of this wave and those which are accompanied by other modifications in the electrical tracing where the prognostic signification is very distinct. Finally, the author takes into consideration the unstable character of this inversion which it is necessary to recognize if one does not wish to make an erroneous prognosis.

Prodger, S. H., and Dennig, H.: A Study of the Circulation in Obesity. J. Clin. Invest. **11**: 789, 1932.

Various phases of the circulation were studied in a group of obese patients who had no demonstrable pathological changes in the circulatory system, chiefly in their

response to mild exercise in the form of walking on a tread mill. The results were compared with the results in a group of normal people. The responses of the cardiac output and arteriovenous oxygen differences to exercise were similar in the two groups. The chief differences between the two groups were found in the vital capacities and in the pulse rates, respiratory rates, blood lactic acid changes, oxygen consumption and oxygen debts in exercise.

On the basis of the findings presented, it is believed that the symptoms which are so commonly associated with cardiac insufficiency and which are frequently observed in cases of simple obesity are not due to an inefficient circulatory response but rather to mechanical and chemical disturbances associated with obesity.

Andrus, E. Cowles, and McEachern, Donald: The Cardiac Manifestations of Hyperthyroidism. Am. J. Med. Sc. **183**: 741, 1932.

In this brief résumé, the authors point out the present conception of the effects of hyperthyroidism upon the heart. They believe that this effect is from its own accelerated metabolism and from the load thrown upon it by the metabolism of the body as a whole.

From the experimental work done on animals, it is believed that the auricles from thyrotoxic animals were more dependent than were the normal controls upon their contemporary oxygen supply which in turn suggested that their rate of oxygen consumption might be increased beyond the normal. It also seemed possible that these preparations were either elaborating lactic acid at the faster rate or were less able to oxidize it or get rid of it by diffusion than the normal auricles. Direct chemical estimations of the lactic acid in cardiac muscle was found to be almost double in the thyrotoxic group as compared with normal rabbits. The glucose and total base was found to have undergone no significant change.

Richards, Dickinson W., Riley, Constance B., and Hiscock, Mabelle: Cardiac Output Following Artificial Pneumothorax in Man. Arch. Int. Med. **49**: 994, 1932.

Studies of the cardiac output and other functions of the circulation have been made before and after artificial pneumothorax in three cases of pulmonary tuberculosis. The cardiac output was decreased following pneumothorax in all cases. Among the other changes that occurred were: decreased vital capacity and residual air and lowered alveolar and blood carbon dioxide levels. The interrelations of some of these changes are discussed.

Bennet, Dudley W., and Kerr, William J.: A Note on Auricular Sounds in a Case of Auricular Flutter. Heart **16**: 109, 1932.

In a patient presenting a disturbed cardiac rhythm resulting from auricular flutter with irregular block, low pitched sound in addition to the usual first and second heart sounds was heard through a binaural stethoscope. The rhythm of the extra sound was regular and rapid, probably about 300 per minute; it was audible at various points over the precordium and most clearly at the third left interspace close to the sternum. The ventricular rhythm was irregular, its average rate being 120 per minute; the first sound was muffled and indistinct while the second was loud and rough. Digital pressure over the left carotid sheath temporarily brought the ventricles to a stand still, the first and second sounds disappearing. During cessation of ventricular contraction the rapid faint ticking sound was still heard, produced apparently by contraction of the auricles.

This interpretation of the adventitious sound was confirmed by recording the electrocardiogram through one of the strings of a double stringed Einthoven galvanometer. The heart sounds were recorded through a sensitive microphone placed on the precordium and electrically connected to a Western Electric stethoscope. The

outgoing wires of the stethoscope were connected to the second string of the Einthoven galvanometer. Records obtained from this procedure are published. From the examination of these records made while ventricular flutter was present and while ventricular action was stopped by vagal pressure, it seems that the sound heard was associated with auricular contraction.

Reid, William D.: The Causation and Propagation of the Heart Beat. New Eng. J. Med. **206**: 1254, 1932.

Data selected from the literature of biochemistry and physiology which form a reasonable explanation of the origin of the heart beat are briefly presented. The author proceeds to discuss these data and emphasizes the lowering of the electrical potential of the heart cells due mainly to the action of potassium irons brought by the blood to the heart.

He discusses the various hypotheses which have been offered to explain the origin of the heart beat in the sinus node rather than in other areas of the heart. This paper presents an interesting short review of these facts as they are known today.

Lewis, Sir Thomas: Raynaud's Disease. New Eng. J. Med. **206**: 1192, 1932.

The author presented a brief summary of his theory as to the mechanism of Raynaud's disease at the meeting of the New England Heart Association. He gave a general idea of experimental work done to support his views. He believes that the well known phenomena associated with this condition are not due to any dysfunction of the vasomotor system but to a "local fault," probably in the arteries of the digits involved. The present article is made out of essential abstracts from an original article published in Heart, volume 15, page 7, 1929.

White, James C.: Raynaud's Disease. New Eng. J. Med. **206**: 1194, 1932.

Six cases of typical Raynaud's disease are reported with observations on the immediate and late effects of sympathetic neurectomy.

Resection of the two upper dorsal ganglia or of the second to fourth lumbar ganglia brought about an immediate paralysis of sympathetic tonus in each case. Whereas, vasomotor paralysis following lumbar sympathectomy has been permanent, the dorsal operation has been followed by a recurrence of sympathetic nerve function in two out of the five cases reported here and of five more not included in this group. Sympathetic nerve activity has reappeared at the end of six months accompanied by the color changes, pain, coldness and ulceration in the tips of the fingers characteristic of Raynaud's disease. When the regenerated vasoconstrictor fibres were again adequately blocked by novocaine or by reoperation, there was a second disappearance of the manifestations of the disease. Therefore, resection of the first and second dorsal sympathetic ganglia alone is insufficient to cause a permanent vasomotor paralysis of the arm. In the recent cases where the operation has been extended upward to include the inferior cervical ganglion, it is hoped that the results will be as permanent as those in the lower extremity.

Advanced stages of the disease with long standing ulceration and sclerodermatous changes may fail to recover completely after vasomotor paralysis. These instances are satisfactorily explained by Lewis' theory of local pathology in the digital vessels.

From the theoretical standpoint, early uncomplicated cases of the disease may be explained either on the basis of Raynaud's original conception of a vasomotor neurosis or by Lewis' theory that normal vasoconstrictor impulses cause the attacks of partial asphyxia by acting on abnormal digital arterioles. Certain objections have been pointed out against each hypothesis. It is, therefore, essential to reserve final judgment until the evidence of one or the other becomes unequivocal.

Levine, Harold D.: Effect of Quinidine Sulphate in Inhibiting Ventricular Fibrillation. Arch. Int. Med. **49**: 808, 1932.

In a series of thirty-six experiments performed on cats, it was found that quinidine sulphate definitely inhibited the facility with which ventricular fibrillation could be produced by cardiac stimulation. It was also found that this effect could not be attributed to manipulation of the heart and the resulting fatigue of the muscle or to the time consumed in the experiments. It is suggested that these results offer a rational background for proper quinidine therapy as a method of preventing sudden death in those conditions in which ventricular fibrillation is prone to occur.

Weisman, S. A.: Auricular Fibrillation. Ambulatory Treatment With Quinidine. Arch. Int. Med. **49**: 728, 1932.

Twenty-four cases of auricular fibrillation who were treated satisfactorily with quinidine by the ambulatory method are reported. Normal rhythm was restored in 17 of these cases.

Of the successfully treated patients, 4 had rheumatic valvular disease, 10 had hypertension, 1 had coronary sclerosis, 1 had diabetes and hypertension, 1 had syphilitic aortitis and 1 had an apparently normal heart. Eighteen of the 24 patients were fifty years of age or over; in 14 of this group, the heart was restored to regular rhythm; in 3 of the remaining 6 patients, regular rhythm was restored. Chronic fibrillation from two months to ten or twelve years had existed in all but one case. Small doses of quinidine were given to start with, following a brief period of the use of tincture of digitalis.

Hyman, Harold T., and Fenichel, Nathan M.: The Management of the Decompensated Cardiac Invalid. Am. J. M. Sc. **183**: 748, 1932.

The records of 100 decompensated cardiac invalids have been summarized and the therapeutic regimen noted. The patients in most instances were victims of rheumatic fever who no longer presented any evidence of activity of heart infection. In the arteriosclerotic group, the acute episodes, such as thrombosis and embolism were part of the past history. The problem was sharply limited to the management of the advanced and decompensated cardiac invalid who had already passed through the active stages of the illness and who had failed to respond to home or ambulatory treatment.

Eight patients were restored to compensation by physiologic rest and dietotherapy. Mechanical removal of fluid from the serous cavities was practiced in 14 of the group. Fifty-six patients were specifically benefited by digitalis and all but 4 in this group had auricular fibrillation.

Sixty-four patients, edematous despite physiologic rest, dietotherapy and digitalization, were relieved by the administration of salyrgan with or without the simultaneous administration of urea and the acid salts. Five patients responded by diuresis to urea alone.

The four cardinal steps in the management of the decompensated cardiac invalid are: (1) physiologic rest and dietotherapy; (2) mechanical evacuation of fluid from the serous cavities; (3) digitalization and (4) the use of diuretics. These four forms of management are discussed.

Hyman, Harold T., and Fenichel, Nathan M.: The Management of the Decompensated Cardiac Invalid. II. Effects of Specific Medication. Am. J. M. Sc. **183**: 753, 1932.

Drug therapy is indicated in the cardiac invalid who remains decompensated despite physiologic rest, dietotherapy and the mechanical evacuation of fluid from

the serous cavities. Digitalis is invaluable in those patients who present auricular fibrillation. Diuretics are equal in importance to digitalis in the management of the decompensated cardiac invalid. Their use is independent of the cardiac rhythm.

Moore, Joseph Earle, Danglade, James H., and Reisinger, John C.: Diagnosis of Syphilitic Aortitis Uncomplicated by Aortic Regurgitation or Aneurysm. Comparison of Clinical and Necropsy Observations in One Hundred and Five Patients. Arch. Int. Med. **49**: 753, 1932.

In the twenty year period from 1910 to 1930, there came to necropsy in the Johns Hopkins Hospital, 105 patients with uncomplicated syphilitic aortitis. The clinical diagnosis of syphilitic aortitis was correctly made during life in 4 of the 105 patients; in 13 more it was suspected that something was wrong with the aorta. On the basis of the symptoms and physical signs recorded, the diagnosis might have been correctly made in 35 additional patients. In 12 instances, the diagnosis was obscured by some other form of cardiovascular disease. Thirty-four patients died with hearts and aortas thought clinically to be normal.

Syphilis was infrequently a major feature of the fatal illness; when it was, aortitis was more often diagnosed correctly than when the final illness was unrelated to syphilis. Hypertension was an infrequent accompaniment of syphilitic aortitis and in spite of the confusion it created in the individual case, its presence did not prevent correct diagnoses. The Wassermann reaction of the blood was positive in 75 per cent of these cases.

The success or failure of the clinical diagnosis is compared with the extent of pathologic change present in the aorta. Symptoms and signs permitting a diagnosis were present in about half of those who showed only relatively slight gross pathologic changes. The criteria of diagnosis are discussed and seven frequently found symptoms or signs listed. The most important are roentgenologic evidence of aortic inhibition, increased retromanubrial dullness, a history of circulatory embarrassment, a change in the tonal quality of aortic second sounds. The other signs less frequently observed are progressive cardiac failure, substernal pain and paroxysmal dyspnea. In patients with proved late syphilis the presence of any three of the seven criteria listed is considered justification for the diagnosis of uncomplicated syphilitic aortitis.

Moore, Joseph Earle, Danglade, James H., and Reisinger, John C.: Treatment of Cardiovascular Syphilis. Results Obtained in Fifty-Three Patients With Aortic Aneurysm and in One Hundred and Twelve With Aortic Regurgitation. Arch. Int. Med. **49**: 879, 1932.

A general consideration of the subject of treatment of cardiovascular syphilis is presented. The material permits some conclusions bearing on important questions of the incidence of cardiovascular syphilis.

An earlier study of the outcome of treatment in early syphilis in the clinic revealed that cardiovascular syphilis developed among these treated patients in inverse ratio to the amount of treatment given early in the infection. Not one of the 117 patients with early syphilis who received three or more courses of arsphenamine and treatment with mercury during periods between the courses, presented any evidence of cardiovascular involvement during the period of observation, while 24 of the 285 patients who had received less than this amount of treatment were observed to acquire syphilitic aortitis, aneurysm or aortic regurgitation. Adequate treatment for early syphilis almost certainly protects the majority of patients so treated against subsequent cardiovascular syphilis.

One hundred and forty-seven of the patients had never received any treatment

for syphilis before the development of cardiovascular syphilis; of the remainder not one had received adequate treatment for early syphilis. Only 4 of the 165 had received arsphenamine at the time of early syphilis and none of these got more than three injections. The evolution of the method of treatment now in use by the authors and subject to minor modifications by many other investigators is outlined. Special stress is laid on sudden death during or immediately following the administration of arsphenamine to patients with syphilitic heart disease, presumably due to ventricular fibrillation; on sudden death from twenty-four to forty-eight hours following an injection due to therapeutic shock and on the therapeutic paradox. The measures taken to avoid these reactions have resulted in the adoption of a method of treatment which is described in detail. This method includes adequate general medical care and the cautious use of mercury, bismuth, the iodides, neoarsphenamine and bismarsen in small doses. All reactions to treatment are meticulously avoided and treatment is prolonged over a period of years.

Using this method of treatment and subdividing the material into four groups on the basis of the amount of treatment given, it is shown that in 22 patients with aortic aneurysm, who received little or no treatment, the mortality during the period of observation was 90 per cent and that the average duration of life from the onset of symptoms to death or, in living patients, to the last observation was nineteen months. In 15 well treated patients with aortic aneurysm the mortality was 40 per cent and the average duration of life seventy-five months. The mortality in 57 patients with aortic regurgitation who received little or no treatment was 91 per cent and the average duration of life thirty months. In 25 well treated patients, the mortality was 16 per cent and the average duration of life seventy-one months. So far as can be judged from the average duration of symptoms, the respective numbers of patients symptom free before and after treatment and the incidence of congestive heart failure before and after treatment in the various treatment groups, the patients in the various groups were approximately similar. That is, it does not appear that the reported deaths occurred only among patients desperately ill before treatment and the reported success only among patients with less serious or less rapidly progressive lesions.

The occurrence of congestive heart failure before or after treatment is of serious prognostic import. In this connection the material is analyzed from several standpoints. Twenty-one of the surviving 56 patients of this series are symptom free and able to work; 26 have some persistent symptoms but can carry on at light work; 9 are incapacitated. Twenty-eight of the 47 still able to work were well treated for syphilis.

Symptomatic relief in cardiovascular syphilis is frequently obtained and its probability is in direct proportion to the amount of treatment given. The occasional alteration of physical signs during or after treatment is discussed. In an analysis from the standpoint of ability to work before and after treatment, well treated patients show up much more favorably than those untreated or badly treated.

Fifty-seven of the 165 patients died of progressive cardiac failure; 28 died suddenly; 11 are dead but the cause of death is unknown; 13 died of some other than cardiovascular syphilis.

The arsenical drugs given to these patients are tabulated and the reactions to them analyzed. From these data the authors conclude that the arsenical drugs of choice are, neoarsphenamine, bismarsen and silver arsphenamine. Old arsphenamine should not be employed in patients with aneurysm or aortic regurgitation. The use of tryparsamide should be limited to patients with complicating neurosyphilis. A fixed positive Wassermann reaction of the blood is the rule in cardiovascular syphilis and the response of this reaction to treatment may be completely disregarded.

Nuzum, Franklin R., and Elliot, Albert H.: Pancreatic Extract in the Treatment of Angina Pectoris and Intermittent Claudication. Arch Int. Med. **49**: 1007, 1932.

An insulin free extract of the pancreas, a vasodilator that modifies the pressor effects of epinephrine and dilates the coronary arteries of the rabbit's heart to a degree exceeding that produced by drugs of the purine group was administered intramuscularly to 20 patients with angina pectoris. Two were not helped, 5 were somewhat relieved, 11 were greatly helped and 2 who were benefited by treatment died later. Forty-one patients with angina pectoris who received the usual treatment and were followed for a like period of time were studied as a control series. In 10 instances no benefit was observed, 14 experienced moderate relief, 14 received pronounced benefit and 3 died. Five patients with intermittent claudication, one with thromboangiitis obliterans and one with cerebral vascular spasms and angina pectoris were benefited to a pronounced degree by treatment with the extract.

Goldring, William, and Chasis, Herbert: Thiocyanate Therapy in Hypertension: II. Its Effect on Blood Pressure. Arch. Int. Med. **49**: 934, 1932.

Data are presented on 50 patients subjected to 74 trials with thiocyanate therapy. In 46 of these patients the hypertension was of essential type and in the remaining 4 patients it was associated with chronic diffuse glomerulonephritis. Forty-four trials were made in the outpatient clinic and thirty with the patients confined to the hospital.

Observations were made on the daily excretion rate of thiocyanate in the urine and the number of days necessary for its complete elimination after continuous medication and after a single dose in patients with essential hypertension and nephritic hypertension and in normal persons. Toxic effects of thiocyanate were not observed in 4 patients with chronic diffuse glomerulonephritis, 3 of whom responded by a satisfactory fall in blood pressure. Therapeutic, toxic or fatal effects could not be anticipated from the amount of the drug administered. The dosage found to be most effective in lowering blood pressure and least often attended by toxic manifestations was 0.326 grams given daily over a period of from fourteen to seventy-eight days. Thiocyanate was 31 per cent effective in lowering blood pressure in this series. Toxic manifestations occurred in 13 patients or 17 per cent of the total group studied.

Miller, H. R., and Feldman, A.: Prolonged Use of Massive Doses of Urea in Cardiac Dropsy. Arch. Int. Med. **49**: 964, 1932.

Urea administered in the way described has been found to be an effective diuretic over long periods of time in selected cases. Whereas, it can function alone over many months in keeping the patient edema free, it may require the supplemental use of other diuretics. This is true for the patient with or without auricular fibrillation. Cases are reported in which its unbroken administration continued over three years. Once the signs of congestive heart failure have been removed urea is valuable in preventing their recurrence. The drug tends to maintain the patients' weight at a low and constant edema free level over months and even years. During such periods the patients often required little or no restrictions of the intake of fluid or salt within ordinary dietary limits.

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